

PDF hosted at the Radboud Repository of the Radboud University Nijmegen

The following full text is a publisher's version.

For additional information about this publication click this link.

<http://hdl.handle.net/2066/146250>

Please be advised that this information was generated on 2018-07-07 and may be subject to change.



ORTHODONTIC

FORCES

AND

TOOTH

MOVEMENT

J.J.G.M. Pilon

Omslag:

Clemens Briels / beeldend kunstenaar

Acryl op linnen

Formaat: 30 x 40 cm

ORTHODONTIC FORCES AND TOOTH MOVEMENT

An experimental study in beagle dogs

ISBN 90-9009878-X

ORTHODONTIC FORCES AND TOOTH MOVEMENT

An experimental study in beagle dogs

Een wetenschappelijke proeve op het gebied van de
Medische Wetenschappen.

PROEFSCHRIFT

ter verkrijging van de graad van doctor
aan de Katholieke Universiteit Nijmegen,
volgens besluit van het College van Decanen
in het openbaar te verdedigen
op dinsdag 1 oktober 1996
des namiddags om 3.30 uur precies

door

Johannes Jacobus Gertrudis Maria Pilon
geboren 12 juni 1959
te Geleen

1996

Druk: ICG printing b.v., Dordrecht

Promotor
Prof.dr. A.M. Kuijpers-Jagtman

Copromotor
Dr. J.C. Maltha

Manuscriptcommissie
Prof.dr. H.H. Renggli
Prof.dr. N.H.J. Creugers
Prof.dr. R. Huiskes

Deze studie werd verricht bij de vakgroep Orthodontie en Orale Biologie (hoofd: Prof.dr. A.M. Kuijpers-Jagtman) van de Katholieke Universiteit Nijmegen.

Dit onderzoek was onderdeel van hoofdprogramma VI "Orale Aandoeningen en Steunweefselziekten".

Contents

Chapter 1	General introduction	13
Chapter 2	Force degradation of orthodontic elastics Submitted to the European Journal of Orthodontics, 1996.	23
Chapter 3	Spontaneous tooth movement following extraction of mandibular third premolars in beagle dogs	39
Chapter 4	Magnitude of orthodontic forces and rate of bodily tooth movement, an experimental study in beagle dogs Published in the American Journal of Orthodontics and Dentofacial Orthopedics (1996) 110: 16-23.	49
Chapter 5	Orthodontic forces and relapse, an experimental study in beagle dogs American Journal of Orthodontics and Dentofacial Orthopedics, 1996. In press.	71
Chapter 6	Histology of periodontal ligament and alveolar bone during bodily orthodontic tooth movement in beagle dogs Submitted to the European Journal of Orthodontics, 1995.	89
Chapter 7	General discussion	113
Chapter 8	Summary	133
Chapter 9	Samenvatting	139
Dankwoord	145
Curriculum vitae	147

Chapter 1

General introduction

1.1 Introduction

The main goal of orthodontic treatment can roughly be described as the improvement of the patient's psycho-social life by enhancing dental and jaw functions and dentofacial aesthetics. More specifically, the goal of orthodontics is to obtain optimal alignment and occlusion of the dentition within the framework of normal functioning and physiological adaptation, acceptable dentofacial aesthetics and self-image, and reasonable stability (Graber and Vanarsdall, 1994). The current definition of orthodontics implies these goals: 'orthodontics/dentofacial orthopedics is the area of dentistry concerned with the supervision, guidance and correction of the growing or mature dentofacial structures, including those conditions that require movement of teeth or correction of malrelationships and malformations of their related structures and the adjustment of relationships between and among teeth and facial bones by the application of forces and/or the stimulation and redirection of functional forces within the craniofacial complex' (American Association of Orthodontists, 1993; Proffit and Fields, 1993).

The four basic treatment modalities orthodontists have at their disposal are, either separately or in combination:

1. repositioning of teeth by orthodontic tooth movement,
2. modifications of dentofacial development by means of orthopaedic modalities,
3. modifications of dentofacial development by means of (myo)-functional forces within and around the oral cavity,
4. combinations of orthodontic and surgical corrections of dentofacial deformities.

Each treatment modality has its own possibilities and limitations. Which one will be the best for a certain malocclusion depends on the type and severity of the anomaly. This has been illustrated by Proffit (1987) with his 'envelope of discrepancy'. Each dentofacial anomaly has three increasing ranges (envelopes) for correction. The inner envelope represents the limits of orthodontic tooth movement alone. The middle envelope shows the range of

correction of combined orthodontic and/or orthopaedic and/or functional therapy. The outer envelope showing the largest range of correction, represents the possibilities of combined orthodontic-surgical treatment.

The dento-alveolar region is the part in which the orthodontist can intervene most directly and with results that are more predictable compared to the results of facial orthopaedics and (myo)-functional therapy. Although tooth movement is the basis of orthodontics, the question how to move teeth most efficiently has still not been answered. The goal of orthodontic therapy is to produce appropriate tooth movement with minimal damage to the root and the surrounding tissues. Only little knowledge exists regarding the relation between applied forces and rate of orthodontic tooth movement. The factors which may influence the rate of tooth movement are largely unknown. Most experiments on tooth movement are limited to a few days and incidently a few weeks. Long term rate of orthodontic tooth movement has never been studied, which is remarkable as an average orthodontic treatment takes about two years, which means that long term characteristics of orthodontic tooth movement are of major importance for clinical orthodontics.

1.2 Biological basis of tooth movement

Orthodontic treatment is based on the principle that force application to a tooth causes remodelling of the periodontal ligament and the bone surrounding the root, which will finally result in displacement of that tooth. Sandstedt reported already in 1904 on bone resorption on the 'pressure' side and bone deposition on the 'tension' side after force application to a tooth. A discussion was soon started about the ideal or optimal magnitude of orthodontic forces. Schwartz stated in 1932 that physiologic tooth movement without damage to the periodontal ligament or the root was possible if the pressure in the periodontal ligament did not exceed capillary blood pressure. According to Stuteville (1938) the magnitude of the orthodontic force was not important as long as its range of activation was less than the width of the periodontal ligament. Reitan advised in 1960 to avoid stress concentrations in the periodontal ligament and advocated low forces and avoidance of tipping

tooth movements. The idea was that with bodily tooth movement forces would be more evenly distributed along the root and the alveolar bone surface (Burstone, 1962). This would reduce the risk of tissue damage and hyalinization, which may impede tooth movement.

The ultrastructural changes in the periodontal ligament have been investigated extensively by Rygh (1973, 1974, 1976, 1992) and Rygh *et al.* (1986). These studies revealed that the normal structure and organization of the periodontal ligament is lost when teeth are moved orthodontically. It has also become clear that degenerative changes are related to force magnitude and the duration of its application. The process of repair and remodelling of periodontal fibres, after hyalinized tissue has been removed by cellular activities, expresses the adaptability of the periodontal ligament. Tooth movement through the alveolar bone is allowed by differentiation and functioning of osteoclastic and osteoblastic cells.

1.3 Tipping versus bodily tooth movement

Up to now, tipping tooth movement has been performed in most experiments. This means that the crown and root of a tooth move at different rates or even in different directions. The rate of movement of the crown and root is dependent of the position of the centre of rotation. This centre of rotation is difficult to determine and it is likely to change during tipping tooth movement (Burstone and Pryputniewicz, 1980). This makes a well defined and reproducible tipping tooth movement almost impossible. Therefore experimental studies with tipping tooth movement are difficult to interpret and the results are very hard to compare with other experiments.

Bodily tooth movement is more suitable for a controlled and reproducible experimental set-up. It can be achieved by using a force acting exactly through the centre of resistance so that the moment to force ratio is zero. The centre of resistance can be calculated for each tooth. However, factors such as root length and the height of the alveolar crest influence the position of this point. Because these measures may change during tooth movement, the centre of resistance may change its position as well, so the

orthodontic force has to be adjusted continuously to avoid tipping tooth movement. Another possibility is a rigid orthodontic appliance that only permits bodily tooth movement. In the present study, this kind of appliance is preferred in order to get reproducible measurements.

1.4 Force magnitude

No consensus exists in the literature on the force magnitude which provides the optimal pressure in the periodontal ligament for orthodontic tooth movement. Schwartz (1932) advocated pressures lower than 2.6 kPa ($1 \text{ kPa} \approx 10 \text{ g/cm}^2$), Jarabak and Fizzell (1963) stated that 20-25 kPa would be ideal for bodily tooth movement. Lee (1965) suggested 18 kPa, and according to Quinn and Yoshikawa (1985) 7-14 kPa would be ideal for canine retraction in humans. The actual force exerted on a tooth during orthodontic treatment is clinically difficult to determine. It is dependent of magnitude, direction, moment to force ratio, duration or frequency, load-deflection curve and range of activation of the total force system. Furthermore the situation is complicated by an interaction between the orthodontic forces and forces from other sources such as muscular activity in rest and during function. In young patients growth of the dentofacial complex is also a factor which has to be taken into account.

The important question of how magnitude of an orthodontic force and the distribution of stress in the periodontal ligament influence the rate of tooth movement, has received little experimental attention. Reitan stated already in 1957: "Very few investigators include a comparison of histologic findings and, on the other hand, the variation in the degree of tooth movement as related to the amount of force applied". This statement is still true and expresses the need for the present study.

1.5 Aim

The purpose of this study is to evaluate the long term relationship between magnitude of orthodontic forces and the rate of bodily tooth movement, to

describe histologic changes in the periodontal ligament and alveolar bone during this period, and to study relapse after active tooth movement.

Beagle dogs were chosen as experimental animals because the structure of their periodontal ligament and alveolar bone, and the shape of the roots of their teeth resemble that of humans (Bartley *et al.*, 1970; Reitan and Kvam, 1971). In our experiments lower second premolars were moved bodily in a distal direction. Three different force levels were used i.e. 50, 100, and 200 cN (1 cN \approx 1 g). The force levels were chosen in relation to the root surface area. The root surface area at the pressure side of the second lower premolar in beagle dogs is estimated to be about 0.5 cm². This means that the experimental forces in clinical terms can be considered as low, medium and heavy forces.

1.6 Preview and organization of this thesis

The chapters that are presented in this thesis represent and deal with the chronological activities prior to and during the actual experiment.

In orthodontics elastics and closing chains are often used to produce forces and move teeth. These products are made in a wide variety with different characteristics. In this experiment elastics have been used to produce orthodontic forces that are continuously present and constant in magnitude. Characteristics as force magnitude and force relaxation of these elastics were measured in different test media and are discussed in chapter 2. At the start of the experiment lower third premolars were extracted. Migration of teeth adjacent to an extraction site may lead to spontaneous closure of the extraction diastema (Stephens, 1983). Chapter 3 deals with these spontaneous tooth movements since they may interfere with the results of the experimental tooth movement, which is planned 6 weeks after the extractions.

In chapter 4 the results of the experimental tooth movement are presented and discussed in relation to the magnitude of the orthodontic force. At the end of the experimental period the orthodontic devices were left in place and the teeth were allowed to relapse. Chapter 5 reports on the process of relapse in relation to the active tooth movement that has been produced

before. The histologic changes in the periodontal ligament during orthodontic tooth movement and relapse are described in chapter 6.

In the general discussion the results of the different parts of this experiment are related to each other and some suggestions are made for future research.

1.7 Literature

- AMERICAN ASSOCIATION OF ORTHODONTISTS (1993). Glossary of dentofacial orthopedic terms. Hester CH (ed). St. Louis (USA): American Association of Orthodontists.
- BARTLEY MH, TAYLOR GN, JEE WS (1970). Teeth and mandible. In: Andersen AC (ed). The beagle as an experimental dog. Ames, Iowa: The Iowa State University Press, pp. 189-215.
- BURSTONE CJ (1962). The biomechanics of tooth movement. In: Kraus BS, Riedel RA (eds). Vistas in orthodontics. Philadelphia (USA): Lea and Febiger, pp. 197-213.
- BURSTONE CJ, PRYPUTNIEWICZ RJ (1980). Holographic determination of centers of rotation produced by orthodontic forces. *Am J Orthod* 77: 396-409.
- GRABER TM, VANARSDALL RL (1994). Orthodontics: current principles and techniques. St. Louis (USA): Mosby-Year Book Inc., pp. 3-19.
- JARABAK JR, FIZZELL JA (1963). Technique and treatment with light-wire appliances, light differential forces in clinical orthodontics. St. Louis (USA): CV Mosby Comp., p. 259.
- LEE BW (1965). Relationship between tooth-movement rate and estimated pressure applied. *J Dent Res* 44: 1053.
- PROFFIT WR (1987). Future of surgical-orthodontic treatment. In: Orthodontics: evaluation and future. Moorrees CFA, Van der Linden FPGM (eds). Alphen aan den Rijn: Samsom Stafleu, pp. 209-218.
- PROFFIT WR, FIELDS HW (1993). Contemporary orthodontics. St. Louis (USA): Mosby-Year Book Inc., pp. 2-16.
- QUINN R, YOSHIKAWA D (1985). A reassessment of force magnitude in orthodontics. *Am J Orthod* 88: 252-260.
- REITAN K (1957). Some factors determining the evaluation of forces in orthodontics. *Am J Orthod* 43: 32-45.

- REITAN K (1960). Tissue behaviour during orthodontic tooth movement. *Am J Orthod* 46 881-900
- REITAN K, KVAM E (1971). Comparative behaviour of human and animal tissue during experimental tooth movement. *Angle Orthod* 41: 1-14
- RYGH P (1973) Ultrastructural changes in the pressure zones of human periodontium incident to orthodontic tooth movement *Acta Odont Scand* 31: 109-122.
- RYGH P (1974) Elimination of hyalinized periodontal tissue associated with orthodontic tooth movement. *Scand J Dent Res* 82. 57-73.
- RYGH P (1976) Ultrastructural changes in tension zones of rat molar periodontium incident to orthodontic movement. *Am J Orthod* 70: 269-281.
- RYGH P (1992) The response of the periodontal ligament to orthodontic forces. In. Bone biodynamics in orthodontic and orthopedic treatment Carlson DS, Goldstein SA (eds) Craniofacial Growth Series Volume 27 Centre for human growth and development Ann Arbor (USA) The University of Michigan
- RYGH P, BOWLING K, HOVLANDSDAL L, WILLIAMS S (1986) Activation of the vascular system a main mediator of periodontal fiber remodelling in orthodontic tooth movement *Am J Orthod* 89 453-468
- SANDSTEDT C (1904) Einige Beitrage zur Theorie der Zahnregulierung *Nord Tandlaek Tidsskr* 5 236-256
- SCHWARTZ AM (1932). Tissue changes incident to tooth movement *Int J Orthod Oral Surg* 18: 331-352
- STEPHENS D (1983) The rate of spontaneous closure at the site of extracted mandibular first premolars *Brit J Orthod* 10 93-97.
- STUTEVILLE O (1938) A summary review of tissue changes incident to tooth movement *Angle Orthod* 8. 1-20

Chapter 2

Force degradation of orthodontic elastics

Jack J.G.M. Pilon
Anne Marie Kuijpers-Jagtman
Jaap C. Maltha

Submitted to the European Journal of Orthodontics, 1996.

2.1 Abstract

Force decay of 6 types of orthodontic elastics was investigated *in vitro*. Elastics were stretched at a fixed distance and initial force levels were measured. They were kept in artificial saliva in the dark, in distilled water in the dark, in air in natural daylight, or in air in the dark. A large variation in initial force levels was found for orthodontic elastics of the same type. Force decay curves showed the same characteristics for all experimental conditions and all elastics. A rapid initial force decay is followed by a period of slow decay. During the first 2 days, elastics showed an average force decay of 13% (range 8-19%) in the "wet" testing media, and an average 5% (range 3-7%) in the "dry" testing media. After 30 days initial differences between the "wet" and "dry" testing media had become smaller. Between 2 and 30 days, elastics stretched in air in natural daylight showed a mean force decay of 14%, which was more than the 9-10% loss in the other experimental groups. Therefore it is advisable to keep stock orthodontic elastics in a dark place. The results indicate that elastics should be stretched prior to clinical use in cases where constant force levels are desired. It is a common practice to use intermaxillary elastics without prestretching and to change them daily. This results in fluctuating forces of unknown magnitude. If force levels are critical, elastics should be measured prior to clinical use. If forces of constant magnitude are desired, elastics should be prestretched in artificial saliva or distilled water for two days. Then they produce almost constant force levels, with a slow loss of force of only 10% during four weeks.

2.2 Introduction

In orthodontics elastics and elastomeric chains are often used to move teeth. Elastics can be made of different materials which may have different characteristics (Wong, 1976; Roff and Scott, 1971). At present polyurethane-based polymers are commonly used whereas in earlier days rubber was more usual. Studies on the load delivered by elastomers were focused on the time dependent behaviour in different test media and the effects of prestretching. The period for which the elastics are used has an effect on the actual force level (Kuster *et al.*, 1986). In load relaxation tests, elastomeric chains showed 16-22% force loss after 3 minutes (von Fraunhofer 1992), and very little additional loss of force thereafter (von Fraunhofer, 1993; Kuster *et al.*, 1986). Also the medium in which the elastics are used influences their behaviour. Force degradation is different in air, water, or in clinical use (Ash and Nikolai, 1978). The loading pattern, which differs from a dynamic functional one, as in intermaxillary use, to a more or less static one, as in space closure, may be another factor influencing force decay. The rate and amount of stretching results in a wide variation in initial load (Hershey and Reynolds, 1975; Kovatch *et al.*, 1976). Slow stretching of elastomeric chains at the moment of insertion results in higher resistance to breakage and less subsequent force decay than fast stretching. This means that regular changing elastomeric chains will result in a force, which works continuously but without constant magnitude. Stretching of elastomeric chains prior to clinical use can result in a more constant force system (Young and Sandrik, 1979). On the other hand, von Fraunhofer (1992) found prestretching elastomeric chains by 100% of the initial length not to affect performance.

Elastics used for intermaxillary traction behave differently from closing chains (Bertl and Droschl, 1986). A specific disadvantage of elastomeric chains is their high degree of stiffness. This means that a small increase in extension causes a great increase in force. For closing interdental spaces clinically often elastic chains are used, which leave few possibilities for individual adaptation to the desired force level.

Most of the literature deals with the behaviour of elastomeric chains. The purpose of this investigation was to determine the following

characteristics of orthodontic elastics when stretched at a fixed distance:

1. Initial force levels.
2. Time-dependent force decay.
3. Influence of experimental conditions (test media).

2.3 Material and methods

Six different types of orthodontic elastics made of polyurethane (Ormco Z-pak elastics, Glendora, Cal., USA) were tested (Table 2-1).

Table 2-1: *The elastics used in this test. Diameter (mm) and force ratings (cN) according to the manufacturer, based on elastics being extended three times the listed diameter.*

Type	Name	Inner Diameter	Force
1	Rabbit	4.8	100
2	Quail	4.8	60
3	Fox	6.4	100
4	Owl	6.4	60
5	Monkey	9.4	100
6	Roadrunner	9.4	60

Acrylic jigs were made to retain the elastics stretched to 2.5 cm. Elastics of all six types were divided into four groups; group A was tested in artificial saliva in the dark, group B was kept in distilled water in the dark, group C was kept in air in natural daylight, and group D in air in the dark. The artificial saliva, prepared according to 's-Gravenmade (1981) contained 32.5 gm bovine mucine/1000 mL and had an ionic composition resembling that of natural saliva. All experiments were carried out at room temperature.

From each type of elastics 20 specimens were tested in each group. Force measurements were made with a calibrated strain gauge at the start of the experiment and after 2, 5, 9, 15, 22, and 30 days. For each type of elastic, mean initial force and remaining force were calculated.

Table 2-2: *Mean initial force F (cN) and SD, and initial force range (cN) for 6 types of elastics when stretched at a distance of 2.5 cm for four experimental conditions;*

A = artificial saliva in the dark, B = distilled water in the dark, C = air in natural daylight, D = air in the dark.

	A		B		C		D		Range
Type	F	SD	F	SD	F	SD	F	SD	
1	150	11	148	11	147	10	148	14	120-190
2	81	7	80	9	80	12	74	10	60-100
3	11	8	108	12	109	7	128	11	70-150
4	53	4	51	4	53	4	77	8	45- 95
5	90	4	81	6	89	6	78	6	65-100
6	35	4	39	6	37	5	43	4	25- 50

The remaining force level was expressed as a percentage of the initial force. Analysis of variance (ANOVA) was used to test differences in behaviour between the elastics (independent of stretching conditions) and differences in stretching conditions (independent of elastics). Force decay as a percentage of the initial force level was compared between the six types of elastics within each experimental condition after 2 and 5 days and differences were tested with Tukey’s multiple range test. Differences between the 6 types of elastics in rate of force decay between 2 and 30 days and between 5 and 30 days were compared within the groups A, B, C, and D with Tukey’s multiple range test.

2.4 Results

Mean force levels of elastics after initial stretching are presented in Table 2-2. A large variation in initial force level was found between elastics of the same type. Deviations ranged from 30% below to 50% above the average force level. In Table 2-3 the remaining force expressed as a percentage of the initial force is listed for the four stretching conditions A, B, C, and D after 2, 5, 9, 15, 22, and 30 days. In Figures 2-1 to 2-4 the

remaining percentages

of force (± 1 SD) are shown for the six types of elastics respectively in group A, B, C, and D as a function of time.

Not only the type of elastics but also the stretching conditions have a significant effect on force value (ANOVA, $p < 0.01$). It was also shown that there was a significant interaction between these two variables (ANOVA, $p < 0.01$). This means that force decay of elastics is not consistent and dependent on the experimental conditions. Therefore it was decided to omit statistically testing of the differences between the four groups of experimental conditions.

Force decay curves show the same characteristics for all experimental conditions (Fig. 2-1 to 2-4). A rapid initial force decay during the first two days is followed by a period of slow decay. This decay was largest in group A and B, ranging from 8-19%, and smallest in group C and D, ranging from 3-7%. The rate of force decay decreased with time for all experimental conditions. After 30 days the initial differences between group A and B on the one hand and C and D on the other hand had become much smaller. Force decay between 2 and 30 days in group A and B ranged from 18-32%, and in group C and D from 18-24%.

Figure 2-1 shows that, after initial force decay, elastics in group A exert an almost constant force from 9 to 30 days.

Figure 2-2 shows that differences in force decay between the 6 types of elastics in group B are larger than in all other groups. This can mainly be attributed to elastics of type 1 and 2.

When Figure 2-3 and 2-4 are compared, it becomes clear that group D is the most homogenous group, whereas in group C elastics of type 1 and 2 shows the largest decrease. Elastics of type 1 and 2 consistently show the largest force decay of 16 and 12% respectively, while mean force decay for types 3-6 are close together (9%).

Table 2-3: *Remaining percentage of force and SD (%) over time for 4 experimental prestretching conditions.*

*A = artificial saliva in the dark, B = distilled water in the dark,
C = air in natural daylight, D = air in the dark.*

Type	Days	A		B		C		D	
		%	SD	%	SD	%	SD	%	SD
		n = 20		n = 20		n = 20		n = 20	
1	2	88	5.4	84	6.3	96	4.1	95	2.9
	5	82	4.2	83	3.5	91	3.5	92	3.6
	9	79	3.8	80	4.1	90	4.0	93	4.1
	15	77	3.8	78	5.0	87	4.1	91	3.2
	22	77	2.9	72	6.3	80	7.4	88	4.4
	30	73	3.4	67	5.6	76	4.9	84	4.0
2	2	83	4.7	81	5.5	93	10.5	96	2.8
	5	80	3.9	82	5.8	88	13.6	92	3.3
	9	77	4.7	78	6.0	89	14.3	93	3.0
	15	75	4.8	72	7.2	86	13.7	91	3.0
	22	74	4.5	70	5.7	79	11.2	88	2.9
	30	73	4.1	67	6.9	76	13.6	88	3.1
3	2	89	6.2	90	3.8	97	5.2	97	1.9
	5	87	4.8	90	4.2	91	4.7	94	2.7
	9	85	4.8	89	5.7	91	5.0	94	2.5
	15	83	5.3	88	4.0	89	6.0	93	2.1
	22	82	5.3	86	3.6	88	6.6	89	4.3
	30	82	5.2	84	5.5	86	6.5	86	4.4
4	2	86	3.4	88	3.2	94	5.3	93	2.9
	5	83	3.5	89	2.8	90	4.9	90	2.8
	9	84	3.4	90	5.0	89	5.4	90	3.4
	15	79	3.8	88	3.9	85	5.0	88	4.2
	22	80	4.2	80	9.0	83	6.1	85	3.2
	30	79	3.8	82	3.9	82	6.1	84	3.7

Type	Days	A		B		C		D	
		%	SD	%	SD	%	SD	%	SD
		n = 20		n = 20		n = 20		n = 20	
5	2	87	4.4	87	2.9	96	5.0	95	1.3
	5	83	4.8	88	4.1	89	5.1	93	1.9
	9	83	4.3	86	5.0	88	4.8	93	2.3
	15	81	5.0	87	5.2	87	5.1	91	1.5
	22	79	4.3	84	4.3	85	5.4	89	2.2
	30	78	4.9	82	3.3	85	5.2	87	1.9
6	2	90	5.1	92	5.7	95	4.5	93	3.4
	5	84	8.4	91	3.8	88	3.7	91	3.9
	9	83	7.2	92	5.8	89	5.0	91	3.2
	15	82	6.5	91	5.7	84	3.9	89	2.9
	22	82	7.1	89	6.1	83	5.3	87	2.2
	30	79	5.6	88	4.6	82	4.5	85	2.8

2.5 Discussion

In this experiment a large variation in initial force level was found between elastics of the same type. Deviations up to 30% below and 50% above the average force level were seen. This means when force levels are critical, measurement of initial force levels prior to clinical use is indicated. According to the manufacturer force levels are based on elastics being extended three times the listed diameter. According to Bales *et al.* (1977), however, the force given on the package was obtained by extending elastics to only twice their diameter. In our experiment elastic type nr. 4 was extended four times which resulted in a mean initial force of 59 cN. According to the manufacturer three times extension would result in 60 cN. Elastic type 6 was extended 2.7 times resulting in 38 cN. According to the manufacturer 3 times extension would result in 60 cN. These data are in contrast to the findings of Bales *et al.* (1977) and show that actual forces are lower than indicated on the package. The other types of elastics forces seem to be more in accordance with the manufacturers indications.

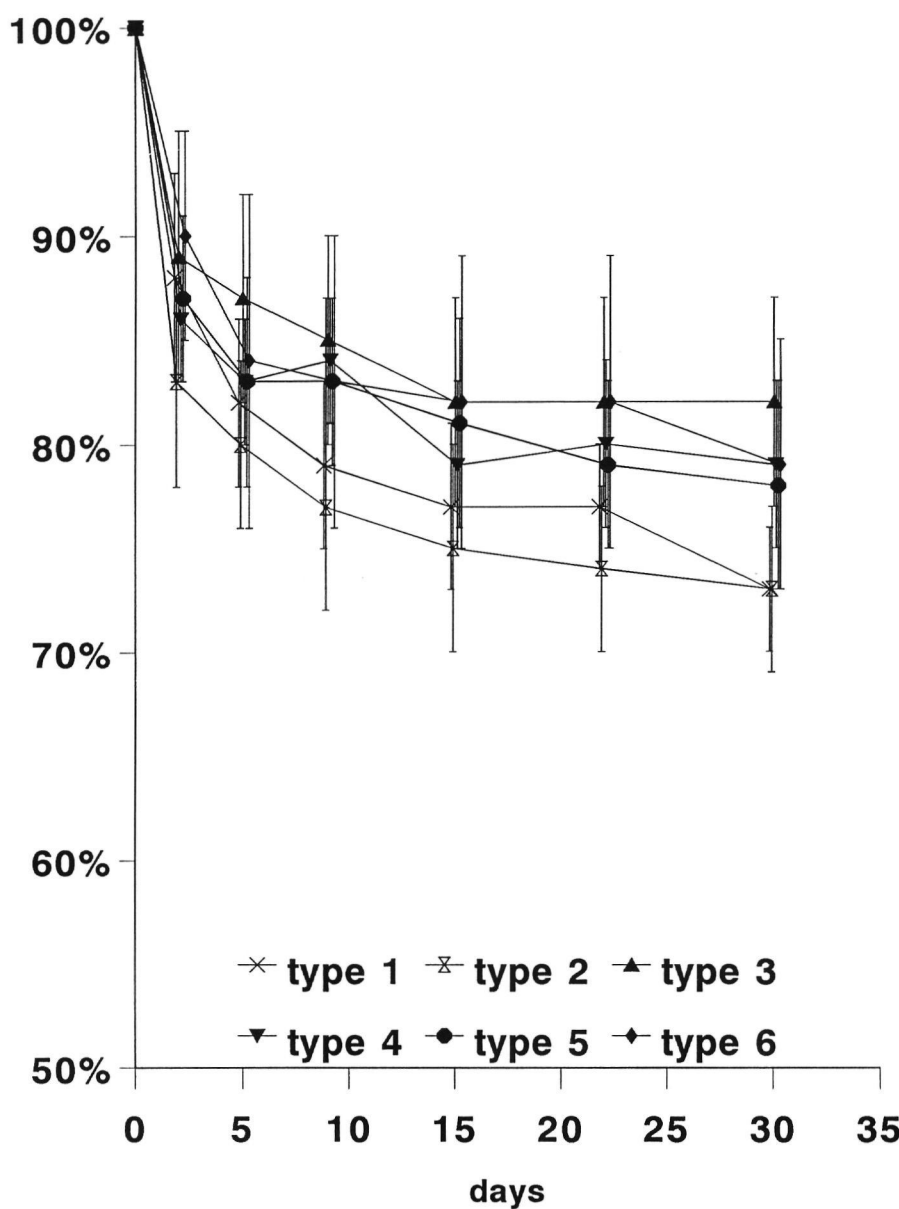


Figure 2-1: Remaining percentage of original force after stretching in artificial saliva in the dark at room temperature (group A).

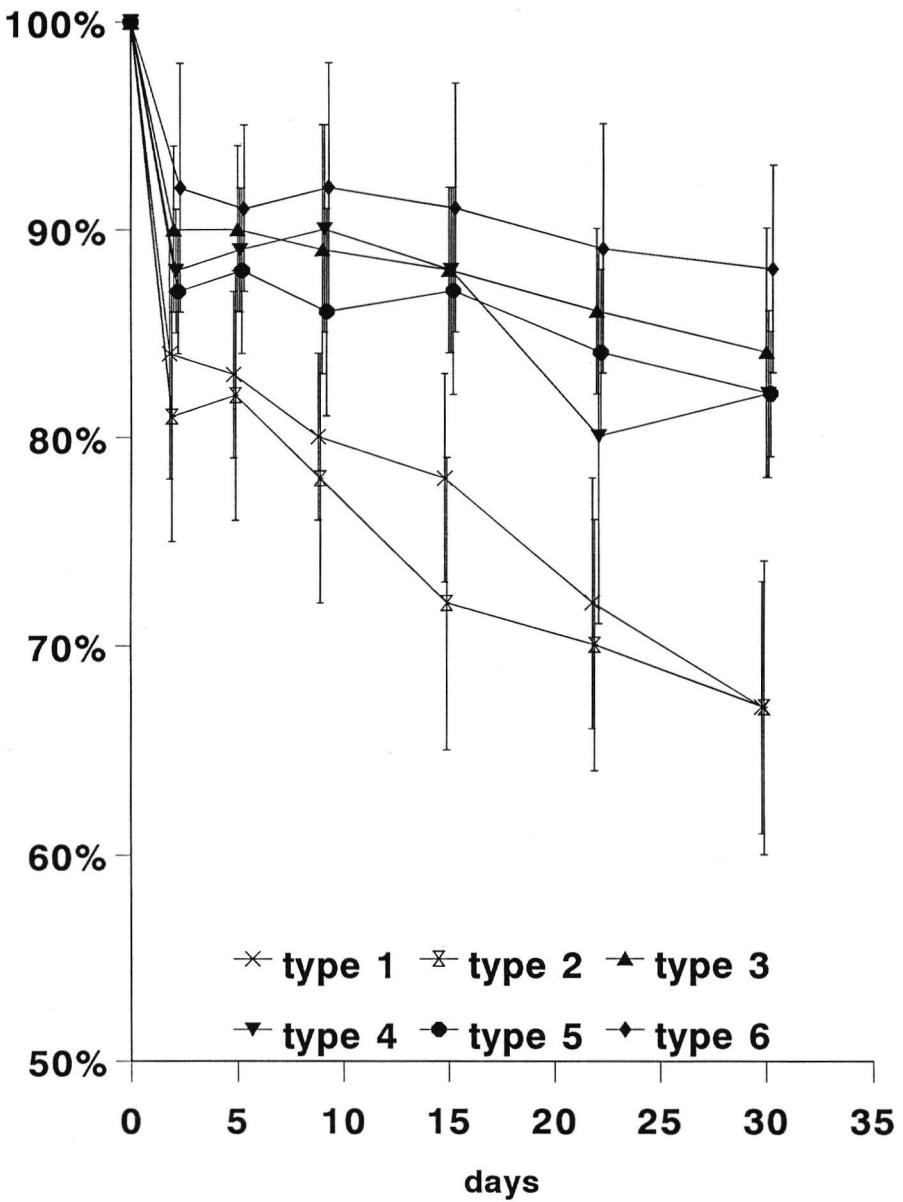


Figure 2-2: *Remaining percentage of original force after stretching in distilled water in the dark at room temperature (group B).*

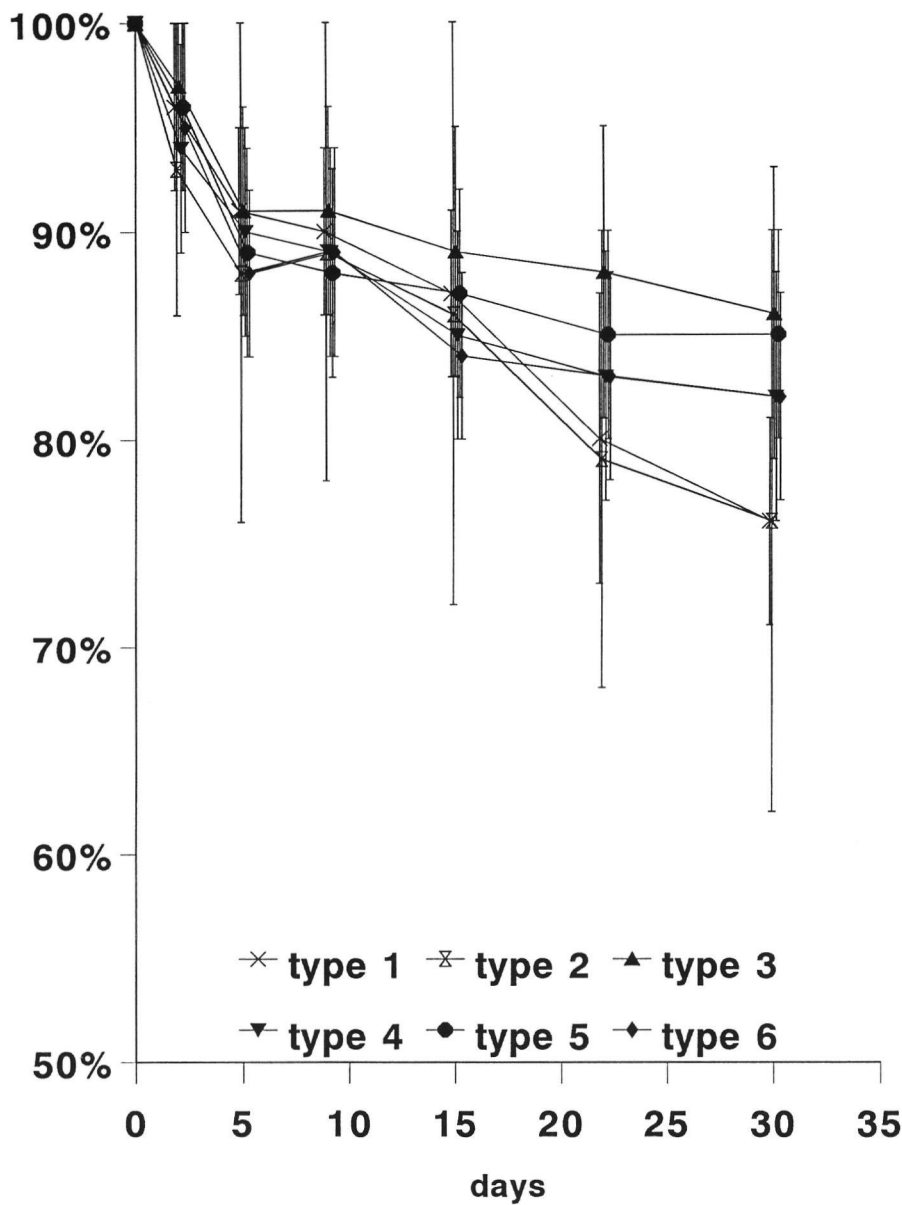


Figure 2-3: *Remaining percentage of original force after stretching in natural daylight in air at room temperature (group C).*

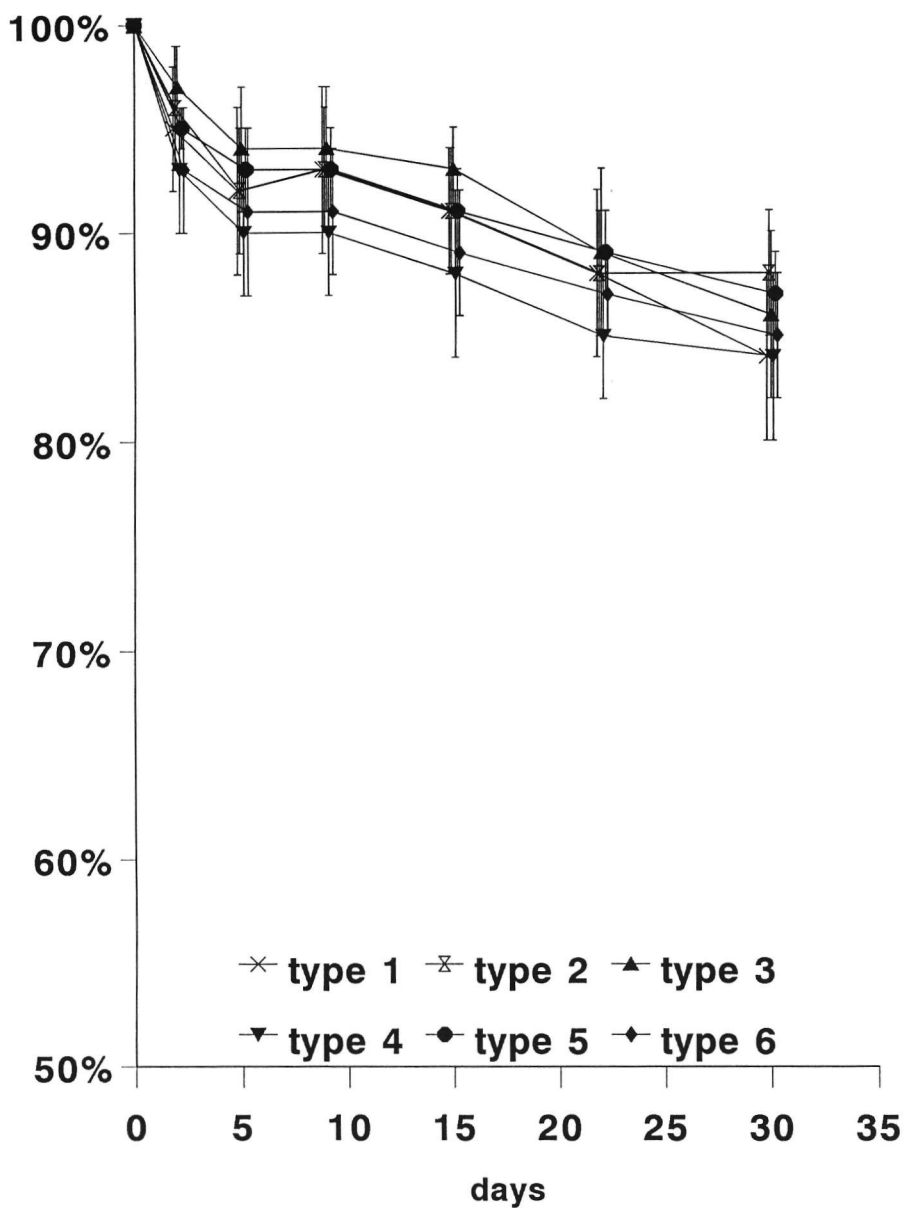


Figure 2-4: *Remaining percentage of original force after stretching in the dark in air at room temperature (group D).*

It was found that stretching for 2 days in artificial saliva as well as in distilled water in the dark causes an average loss of 13% of the initial tension. Stretching in air in the daylight as well as in the dark causes 5% force decay in the same period. There is a more or less continuous loss of force throughout the experimental period in the "dry" testing media, while in the "wet" testing media rapid initial force decay is followed by a period of smaller loss of force. However, differences in force decay in the four testing media between 2 and 30 days are small. In the presence of daylight, however, mean force decay was 14%, which was larger than in the other experimental groups (9-10%).

Ash and Nikolai (1978) found no significant differences in relaxation of elastomeric chains *in vivo* versus simulation in a water-bath after 1 week of continuous loading. After 3 weeks, a higher percentage of force degradation was found *in vivo*. They also found that in-air experiments led to significant less loss of force compared to a water-bath. The time dependent behaviour seems to be related to the amount of stretching. Elastics of type 1 and 2, which have the smallest passive diameter, show the largest force decay in all groups of 16 and 12% respectively between 2 and 30 days, while the mean for elastics 3-6 is 9%.

Elastomeric chains are found to have an even larger force decay than elastics (Hershey and Reynolds, 1975; De Genova *et al.*, 1985; Rock *et al.*, 1986). Several methods have been proposed to overcome initial loss of force in clinical use. Elastomeric chains, which are stretched in distilled water for a period of 1 day or 3 weeks prior to clinical use, exert 80 or 92% respectively of their initial force level for three weeks in the mouth (Brantley *et al.*, 1979). Quick stretching of elastomeric chains before use does not reduce force decay (Young and Sandrik, 1979). Because of the initial loss of force, elastomeric chains have to be overstretched at the moment of insertion. When overstretching 300-400%, the elastic limit can be exceeded so in reality the effective force could be decreased (Andreasen and Bishara, 1970). Stevenson and Kusy (1994) advise to prestretch elastomeric chains to a 50% increase in length for one minute. They also found an increase in temperature to significantly influence the deterioration of the mechanical properties of elastomeric chains.

As is generally accepted, high initial loads in orthodontic treatment can lead to extensive damage of the periodontal ligament and the subsequent hyalinization can impede tooth movement (Rygh, 1973). From this point of view orthodontic elastics are a better choice for producing tooth movement than unconditioned elastomeric chains. Initial force decay of elastics is smaller and precise force levels are easier to obtain because elastics have a smaller degree of stiffness. When elastics are stretched prior to clinical use, not only high initial force levels can be avoided, but also an almost constant force level can be produced for several weeks. It should be kept in mind, that these results are obtained from elastics stretched at a fixed distance. The time dependent behaviour during a dynamic loading pattern, as in intermaxillary use, may be different.

In clinical orthodontics, no strict rules apply to the most effective force magnitude for intermaxillary elastics, but the common practice of using elastics without prestretching, and changing them daily, results in fluctuating forces of unknown magnitude. From a biological point of view, the question of the most effective force magnitude is still unanswered, but in order to apply orthodontic forces with elastics in a controlled way, it is advisable to prestretch them prior to use and, ideally, to measure the force they exert prior to insertion. After prestretching for two days in distilled water or artificial saliva, orthodontic elastics can exert an almost constant force for at least four weeks.

2.6 Literature

- ANDREASEN GF, BISHARA SE (1970). Comparison of alastik chains with elastics involved with intra-arch molar to molar forces. *Angle Orthod* 40: 151-158.
- ASH JL, NIKOLAI RJ (1978). Relaxation of orthodontic elastomeric chains and modules in vitro and in vivo. *J Dent Res* 57: 685-690.
- BALES TR, CHACONAS SJ, CAPUTO AA (1977). Force-extension characteristics of orthodontic elastics. *Am J Orthod* 72: 296-302.
- BERTL WH, DROSCHL H (1986). Forces produced by orthodontic elastics as a function of time and distance extended. *Eur J Orthod* 8: 198-201.

- BRANTLEY WA, SALANDER S, MYERS CL, WINDERS RV (1979). Effects of prestretching on force degradation characteristics of plastic modules. *Angle Orthod* 49: 37-43.
- DE GENOVA D, McINNES-LEDOUX P, WEINBERG R, SHAYE R (1985). Force degradation of orthodontic elastomeric chains - A product comparison study. *Am J Orthod* 87: 377-384.
- 'S-GRAVENMADE EJ (1981). Clinical applications of saliva substitutes. *Front Oral Physiol* 3: 154-161.
- HERSHEY HG, REYNOLDS WG (1975). The plastic module as an orthodontic tooth moving mechanism. *Am J Orthod* 67: 554-562.
- KOVATCH JS, LAUTENSCHLAGER EP, APFEL DA, KELLER JC (1976). Load-extension-time behaviour of orthodontic alastics. *J Dent Res* 55: 783-786.
- KUSTER R, INGERVALL B, BURGIN W (1986). Laboratory and intra-oral tests of the degradation of elastic chains. *Eur J Orthod* 8: 202-208.
- ROCK WP, WILSON HJ, FISHER SE (1986). Force reduction of orthodontic elastomeric chains after one month in the mouth. *Brit J Orthod* 13: 147-150.
- ROFF WJ, SCOTT JR (1971). Fibers, films, plastics and rubbers. In: *Handbook of common polymers*. London: Butterworths.
- RYGH P (1973). Ultrastructural changes in pressure zones of human periodontium incident to orthodontic tooth movement. *Acta Odontol Scand* 31: 109-122.
- STEVENSON JS, KUSY RP (1994). Force application and decay characteristics of untreated and treated polyurethane elastomeric chains. *Angle Orthod* 64: 455-467.
- VON FRAUNHOFER JA (1992). The effects of artificial saliva and topical fluoride treatments on the degradation of the elastic properties of orthodontic chains. *Angle Orthod* 62: 265-274.
- VON FRAUNHOFER JA (1993). Stress relaxation of polyurethane elastomers. *Procs 22nd Conf of NATAS*: 542-547.
- WONG AK (1976). Orthodontic elastic materials. *Angle Orthod* 46: 196-205.
- YOUNG J, SANDRIK JL (1979). The influence of preloading on stress relaxation of orthodontic polymers. *Angle Orthod* 49: 104-109.

Chapter 3

Spontaneous tooth movement following extraction of mandibular third premolars in beagle dogs

3.1 Abstract

Spontaneous closure of an extraction diastema is often seen in human individuals. The rate and degree of this type of tooth movement may be influenced by wound healing, scar tissue formation, and bone metabolism. Also functional and occlusal forces, and relief of crowding may be involved. In our experiments on tooth movement in beagle dogs, spontaneous movements after extraction of a mandibular premolar could bias the estimation of the rate of active tooth movement. Therefore the aim of the study presented in this chapter was to evaluate spontaneous tooth movement after extraction in a situation where neither occlusion nor interproximal contacts were present, as is the case in the mandibular premolar region in beagle dogs.

Eighteen young adult male beagle dogs were used in this experiment. Alginate impressions of the mandibular arch were taken before extraction of the lower third premolars and 4, 8, and 12 weeks thereafter. All dogs had a complete permanent dentition at the start of the experiment. On the dental casts the position of the crown tips of the canine, first, second and fourth premolar, and the first molar were digitized with an electronic measuring device. Changes in distances between these teeth were calculated. A continuous increase of nearly all interdental distances was found. This may be explained by growth of the mandible with drifting of teeth or by eruptive movements of mandibular teeth, which have diverging inclinations.

3.2 Introduction

Spontaneous closure of extraction diastema's is a common experience in dental practice. The rate of spontaneous tooth movement after extraction shows large individual differences, which may be attributed to the reactions in the periodontal ligament (Prösl and Kröncke, 1979). The rate of space closure was found to be most rapid immediately after tooth removal and consistently greater in males than females (Stephens, 1983). The phenomenon of spontaneous space closure is not restricted to humans. Varying degrees of spontaneous migration of teeth towards the extraction diastema after the removal of deciduous molars have been reported also in monkeys (Richardson, 1965). Pietrokovski (1970), however, found in his experiments in monkeys only mesial drift, not only distal but also mesial from extraction sites.

The reason for spontaneous migration of teeth after extractions is still unclear. If one of the factors which maintain an equilibrium in the tooth position changes, pathologic migration may be the result (Glickman, 1979). Extraction of a neighbouring tooth is such a factor. Other important factors may be the relief of crowding (Cryer, 1965) or functional aspects, as Cookson (1971) found a strong correlation between space closure after extraction and a competent lip seal.

Forces originating from occlusion and interdigitation may also be involved in the process. According to Van Beek and Fidler (1977), interdental spaces close faster when the opposing teeth are in occlusion. Occlusal forces have a mesial component, but according to Moss and Picton (1967) this is not responsible for mesial drift in monkeys. From another experiment Moss and Picton (1970) concluded that both forces from occlusion as well as forces from the cheeks and the tongue do not play a significant role in mesial drift in monkeys. Other factors than forces from biting and occlusion, especially contraction of the transseptal fibre system, may play a dominant role in approximal drifting of teeth (Picton and Moss, 1979). Finally, the presence or absence of third molars might play a role in the spontaneous closure of extraction diastema's (Cookson, 1971).

The present study was part of a larger experiment in which the

relationship between force magnitude and rate of orthodontic tooth movement was determined. For these experiments, which are described in the next chapters of this thesis, lower third premolars were extracted and second premolars were moved distally. Spontaneous tooth movements after extraction, however, may eventually lead to an over- or underestimation of the rate of tooth movement. Therefore it was necessary to get more insight into spontaneous migration after extraction.

3.3 Subjects and methods

Eighteen young adult male beagle dogs aging between 7 and 12 months (mean 9.3 months) with a complete permanent dentition were used as experimental animals in this study. After premedication with 1.5 mL Thalamonal^R (Fentanyl 0.05 mg/mL and Droperidol 2.5 mg/mL; Janssen Pharmaceutica, Beerse, Belgium) and subsequent general anaesthesia with 15 mg/kg Nesdonal^R (Thiopental Sodium 50 mg/mL; Rhone-Poulenc Pharma, Amstelveen, The Netherlands), the lower third premolars on both sides were removed after hemisection. Careful operation was indicated to prevent excessive resorption of the residual ridge. Alginate impressions (CA 37, Cavex B.V. Haarlem, The Netherlands) of the lower dental arches were taken at the start of the experiment prior to the extraction and after 4, 8, and 12 weeks. Therefore the dogs were sedated with 3 mL of a generic preparation containing 10 mg Oxycodon HCl, 1 mg Acepromazine, and 0.5 mg Atropine Sulphate per mL. Stone casts were made (Silkyrock Violet, Whiphmix Corporation, Louisville, Kentucky, USA).

The positions of the tips of the canine, the first, second, and fourth premolar and the first molar on each cast were digitized with an electronic measuring device for dental casts (Van der Linden et al, 1972) by two independent observers. These points were projected on the line connecting the tips of the canine and first molar. Distances between all the projection points were calculated (Fig. 3-1). Measurements for the left and right side were pooled and the means and SEMs were calculated.

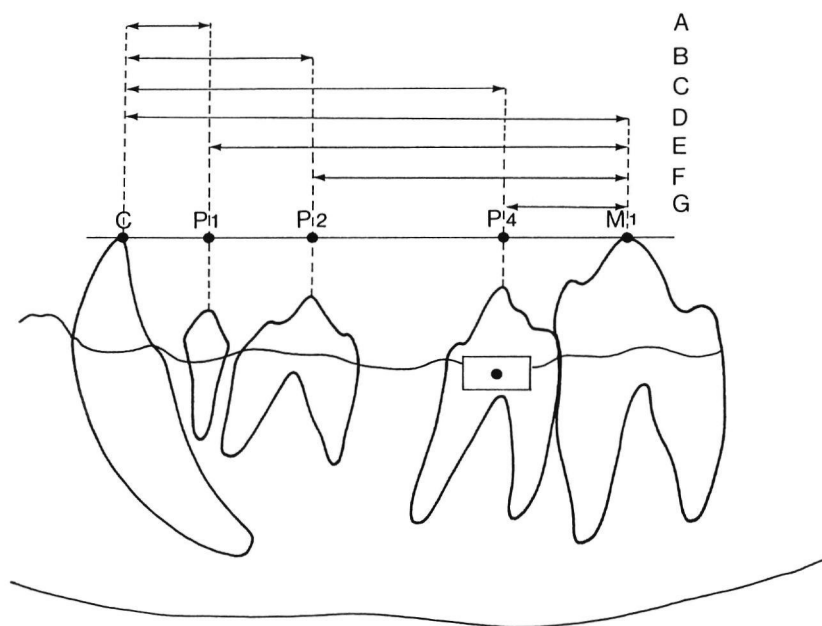


Figure 3-1: *Definition of the distances A-G.*

- A: canine to first premolar projection*
- B: canine to second premolar projection*
- C: canine to fourth premolar projection*
- D: canine to first molar*
- E: first premolar projection to first molar*
- F: second premolar projection to first molar*
- G: fourth premolar projection to first molar*

Intra- and inter-observer agreement was assessed by calculating differences of the means of the distances A to G within and between the two observers for 11 models using Student's t-test. Mesio-distal migrations were calculated as the changes in distance between the projection points from the start of the experiment to 4, 8, and 12 weeks afterwards. The change over time of each of the parameters was studied with Student's t-test.

3.4 Results

The extraction wounds epithelialized rapidly and healing took place without any complication. After 4 weeks the dental cast of one dog was missing. Between 8 and 12 weeks after the start of this experiment 7 dogs were killed for histological evaluation, while the dental cast of one dog was missing.

Intra- and inter-observer differences for the measurements on dental casts were not significant. The total error of the measurements was 0.26 mm.

Table 3-1: *Mean changes (ΔX) and SEM in mm for distances A to G from 0 to 4, 0 to 8, and 0 to 12 weeks after extraction. Significance level for the differences is indicated as follows:*

NS = not significant

** = significance level $0.01 < p \leq 0.05$*

*** = significance level $p \leq 0.01$.*

Distance	0-4 weeks n = 17			0-8 weeks n = 18			0-12 weeks n = 9		
	ΔX	SEM		ΔX	SEM		ΔX	SEM	
A	0.34	0.11	**	0.28	0.15	NS	0.79	0.17	**
B	0.37	0.12	**	0.55	0.17	**	1.22	0.20	**
C	0.48	0.10	**	0.65	0.14	**	1.37	0.23	**
D	0.64	0.21	**	0.95	0.18	**	1.52	0.21	**
E	0.31	0.18	NS	0.66	0.14	**	0.73	0.18	*
F	0.28	0.19	NS	0.40	0.09	**	0.28	0.18	NS
G	0.16	0.16	NS	0.29	0.11	**	0.15	0.15	NS

The mean changes for distances A to G during 4, 8, and 12 weeks after extraction are listed in Table 3-1. After 12 weeks the changes in distance from the projections of the first, second and fourth premolar to the canine are 1.1, 4.4, respectively 9.1 times as large as the changes to the first molar. The total distance D between canine and first molar increased continuously.

The distance between the second premolar and the first molar (F) and the distance between the fourth premolar and the first molar (G), was not significantly changed after 12 weeks.

3.5 Discussion

This experiment was performed in beagle dogs, because they took part in a larger study on the relationship between the magnitude of orthodontic forces and the rate of tooth movement. Spontaneous migrations after extraction of the third premolar in the lower jaw could bias the estimation of the rate of active orthodontic tooth movement. Two of the factors that may be related to the rate of spontaneous migrations after extractions are crowding and occlusal interference. Because of the natural presence in beagle dogs of spacing in the lower jaw from canine to first molar and the presence of an open bite in this region these factors are excluded.

It became clear that the largest changes were found in the distances in which the canine was involved. The increase in the distance between the canine and the first premolar causes approximately half of the total increase between canine and first molar. The further a tooth is positioned away from the canine, the larger is the increase in their distance over time. Increases related to the canine are much larger than increases related to the first molar. The distance between the second and fourth premolar and the first molar was not significantly changed in the first 12 weeks after extraction. The results indicate a process of constant diverging movement between the canine and the first molar in young adult beagle dogs. The amount of tooth movement depends on the position in the dental arch and seems to be related to the distance between the canine and that tooth.

The distance between the two teeth adjacent to the extraction site does not decrease. Functional aspects might be involved as in beagle dogs the tongue is normally positioned between the upper and lower premolars resting in the open bite region, while the tonus of the buccal musculature is very low.

The fact that all teeth are migrating away from each other could be

explained in two ways. In the young adult beagle dogs growth of the lower jaw may provide additional space for the teeth to spread out. This could be in contrast to the finding in humans that teeth move in a mesial direction during the development of the dentition and that, if a tooth is extracted, the distal teeth will move forward (Salzmann, 1938; Friel, 1945). Brodie (1934) noted the mesial inclination of molars and the distal inclination of premolars in herbivores. If teeth with these inclinations erupt, the direction of eruption may explain the direction of drifting of teeth, as was also noted by Moss and Picton (1967). The inclination of the lower canine in beagle dogs is to the mesial and of the fourth premolar and first molar slightly to the distal, so further eruption of teeth with their divergent inclinations may cause an increase in distance between the crown tips. It is formerly shown by Maltha (1982) that eruption of teeth in beagles at least continues until the age of 14 months. The mean age of the beagle dogs at the start of this experiment was 9.3 months, so the process of eruption was not yet completed.

For the experiments, described in the next chapters, the divergent eruption pattern of the lower canine and first molar is of no significance because the distance between these teeth is fixed with an anchorage bar. No significant change was found in the distance between the second and fourth premolar, both adjacent to the extraction site, and the first molar in the first 12 weeks after extraction of the third premolar. Therefore it is concluded that spontaneous migration is not a factor of importance in our experiments on tooth movement.

3.6 Literature

- BEEK VAN H, FIDLER VJ (1977). An experimental study of the effect of functional occlusion on mesial tooth migration in Macaque monkeys. *Archs oral Biol* 22: 269-271.
- BRODIE AG (1934). The significance of tooth form. *Angle Orthod* 4: 335-350.
- COOKSON A (1971). Space closure following loss of lower first premolars. *Dent Practit* 21: 411-416.
- CRYER BS (1965). Lower arch changes during the early teens. *Trans Eur Orthod Soc*: 87-99.

- FRIEL ES (1945). Migrations of teeth following extraction. *Proc Roy Soc Med* 38: 456-462.
- GLICKMAN I (1979). Pathologic migration; tooth mobility. In: Glickman's Clinical Periodontology, 5th ed. Philadelphia (USA): WB Saunders, pp. 294-302.
- LINDEN VAN DER FPGM, BOERSMA H, ZELDERS T, PETERS KA, RABEN JH (1972). Three-dimensional analysis of dental casts by means of the Optocom. *J Dent Res* 51: 1100.
- MALTHA JC (1982). The process of tooth eruption in beagle dogs. PhD Thesis, University of Nijmegen, The Netherlands.
- MOSS JP, PICTON DCA (1967). Experimental mesial drift in adult monkeys (*Macaca irus*). *Archs Oral Biol* 12: 1313-1320.
- MOSS JP, PICTON DCA (1970). Mesial drift of teeth in adult monkeys (*Macaca irus*) when forces from the cheeks and tongue had been eliminated. *Archs Oral Biol* 15: 979-986.
- PICTON DCA, MOSS JP (1979). The effect on approximal drift of altering the horizontal component of biting force in *Macaca irus* monkeys. *J Dent Res* 58 (Special Issue C): 1253.
- PIETROKOVSKI J (1970). Tooth drift and changes in the temporomandibular joint following tooth extraction in the monkey. *J Period* 41: 353-358.
- PRÖSL R, KRÖNCKE A (1979). Über die Wanderungsgeschwindigkeit lückenbenachbarter Zähne. *Dtsch Zahnärztl Z* 34: 234-235.
- RICHARDSON ME (1965). The direction of tooth movement subsequent to the extraction of teeth in the rhesus monkey. *Rep Congr Eur Orthod Soc* 41: 133-151.
- SALZMANN JA (1938). A study of orthodontic and facial changes, and effects on dentition attending the loss of first molars in five hundred adolescents. *Am Dent Ass J Dent Cosmos* 1: 892-905.
- STEPHENS CD (1983). The rate of spontaneous closure at the site of extracted mandibular first premolars. *Br J Orthod* 10: 93-97.

Chapter 4

Magnitude of orthodontic forces and rate of bodily tooth movement, an experimental study in beagle dogs

Jack J.G.M. Pilon
Anne M. Kuijpers-Jagtman
Jaap C. Maltha

Published in the American Journal of Orthodontics and Dentofacial Orthopedics (1996) 110: 16-23.

4.1 Abstract

The relationship between the magnitude of a constant continuous orthodontic force and the rate of bodily tooth movement was studied. In 25 young adult male beagle dogs lower third premolars were extracted and bone markers were implanted in the mandible. Sixteen weeks later, an orthodontic appliance was placed and elastics exerting 50, 100, or 200 cN were attached to the second lower premolar to produce bodily distalization. In each dog different forces were used at the left and the right side. As a control group on 8 sides orthodontic appliances were placed without elastics. Tooth movement was measured directly with a digital calliper twice a week during 16 weeks. Resulting curves could be divided in four phases. Large individual differences were found in the rate of tooth movement. Tooth movement at the left and right side of each dog, however, were highly correlated. No significant differences in the duration of each phase nor in the mean rate of tooth movement during each phase were found between the three force groups. Maximum rate of tooth movement was about 2.5 mm per month in all force groups. There were no significant differences in the mesial movement of the anchorage unit between the force groups. It is concluded that under the circumstances of this study magnitude of force is not decisive in determining the rate of bodily tooth movement, but individual characteristics are.

4.2 Introduction

In orthodontics no consensus exists on how to move teeth most efficiently. An optimal approach should result in the highest possible rate of tooth movement without irreversible damage to the periodontal ligament, the alveolar bone, or the root. The formation of cell-free areas in the periodontal ligament cannot be avoided, even with light forces¹. Hyalinization occurs less frequently during bodily than during tipping movements², because forces are more evenly distributed along the root surface during bodily movement.

Large individual variations in the rate of bodily tooth movement were found in experiments on cats, after application of identical forces³. These differences could be attributed to bone density, supra-alveolar fibres, structure of collagen fibres, and cellular activity in the periodontal ligament^{3,4}. It was suggested that an optimal force range for tooth movement exists, related to the root surface area⁵. Most interesting is the relationship between magnitude of pressure in the periodontal ligament and the rate of tooth movement. A linear relationship may exist up to a maximum rate of tooth movement which cannot be increased by further increase of force^{6,7}.

Experimental studies on tooth movement are often difficult to interpret because the description of orthodontic forces is not uniform, and incomplete. Another problem is that in earlier studies particularly tipping movements were investigated^{7,8,9}, where the tooth crown is used as reference point for measuring tooth movement. The results of these studies are hard to interpret because the relation between the rate of crown and root movement is dependent on the position of the centre of rotation which is difficult to determine and probably changes during tipping tooth movement¹⁰.

Not only is the question of the relationship between the force magnitude per unit of root surface area and rate of tooth movement not answered, but equally important is a reliable registration of time-displacement curves of orthodontic tooth movement over a period longer than 4 weeks. This has never been done. The purpose of this experiment is to study the relationship between the magnitude of a constant and continuous orthodontic force and the rate of bodily tooth movement during a period of 16 weeks.

4.3 Materials and methods

4.3.1 Experimental set-up

A group of 25 young adult male beagle dogs was used including 8 pairs of twin brothers and one triplet. The age of the dogs varied between 1 and 1.5 years. After extraction of the mandibular third premolars bodily distalization of the lower second premolars was produced. The sides were divided over four groups: control sides ($n = 7$) in which the appliance was placed and no force was exerted and three experimental groups of sides in which 50 cN ($n = 16$), 100 cN ($n = 14$), and 200 cN ($n = 14$) was applied. In total 44 sides were present in the experimental groups: one former control side (0 cN) was added to the 50 cN group, one to the 200 cN group, and at one side the 200 cN appliance was lost. At the left and right side of each dog different forces were used which were selected at random (Table 4-1).

Table 4-1: *Number of dogs (n) with the forces (cN) applied to the second premolar at the left and right side. Forces were selected at random for the left and right side.*

n	Force	
	side 1	side 2
3	0	50
2	0	100
2	0	200
6	50	100
6	50	200
6	100	200

4.3.2 Surgical procedures

The dogs were premedicated with 1.5 mL Thalamonal^R (fentanyl 0.05 mg/mL and droperidol 2.5 mg/mL; Janssen Pharmaceutica, Beerse, Belgium) and anaesthetized with Nesdonal^R 15 mg/kg (thiopental sodium 50 mg/mL; Rhone-Poulenc Pharma, Amstelveen, the Netherlands). The lower left and right third premolars were extracted after hemisection. Prior to the start of

the experimental tooth movement, under general aesthesia, on both sides of the mandible three tantalum bone markers (Ole Dich, Hvidovre, Denmark) were implanted according to the method of Björk¹¹.

4.3.3 *Orthodontic procedure*

Sixteen weeks after extraction of the third premolars alginate impressions (CA 37, Cavex Holland BV, Haarlem, The Netherlands) of the lower dental arches were made after sedation with 1 mL of a generic preparation containing 10 mg oxycodon HCl, 1 mg acepromazine, and 0.5 mg atropine sulfate per mL, which was injected subcutaneous. The impressions were poured out in stone (Silkyrock Violet, Whipmix Corporation, Louisville, Kentucky, USA) within a few hours. On the dental casts an appliance was constructed allowing bodily distalization of the second premolar (Fig. 4-1). Crowns were modelled in wax on the second and fourth premolar and onlays on the distal concave surface of the canine and the lingual surface of the permanent first molar. The canine, fourth premolar and first molar were connected with a lingual bar to serve as an anchorage unit. The appliance was casted in a chrome-cobalt alloy (Wironium Bego, Bremen, Germany) and the inner surfaces were etched to improve retention. At the buccal side of the second premolar crown a rigid sliding wire (316 Chrome-Nickel stainless steel) with a diameter of 2 mm was soldered. At the buccal side of the fourth premolar crown a ring was modelled into which a round steel tube was glued (Uhu epoxy glue, Beecham, Milan, Italy). The inner diameter of the tube was 0.02 mm larger than the sliding wire at the second premolar. The mesial end of the sliding wire at the second premolar and the distal side of the buccal tube at the fourth premolar were prepared plan-parallel. They were used as reference planes for the measuring procedure.

Under general aesthesia, the enamel was polished and etched and the appliances were cemented with Panavia Ex Dental Adhesive (Cavex Holland BV, Haarlem, The Netherlands) at the left and right side of each dog. During the experimental period of 16 weeks, elastics (Ormco Z-pak elastics, Glendora, Cal., USA) were attached from a buccal hook on the second premolar crown to a buccal hook on the fourth premolar crown. Because elastics show a great loss of force after initial extension they were

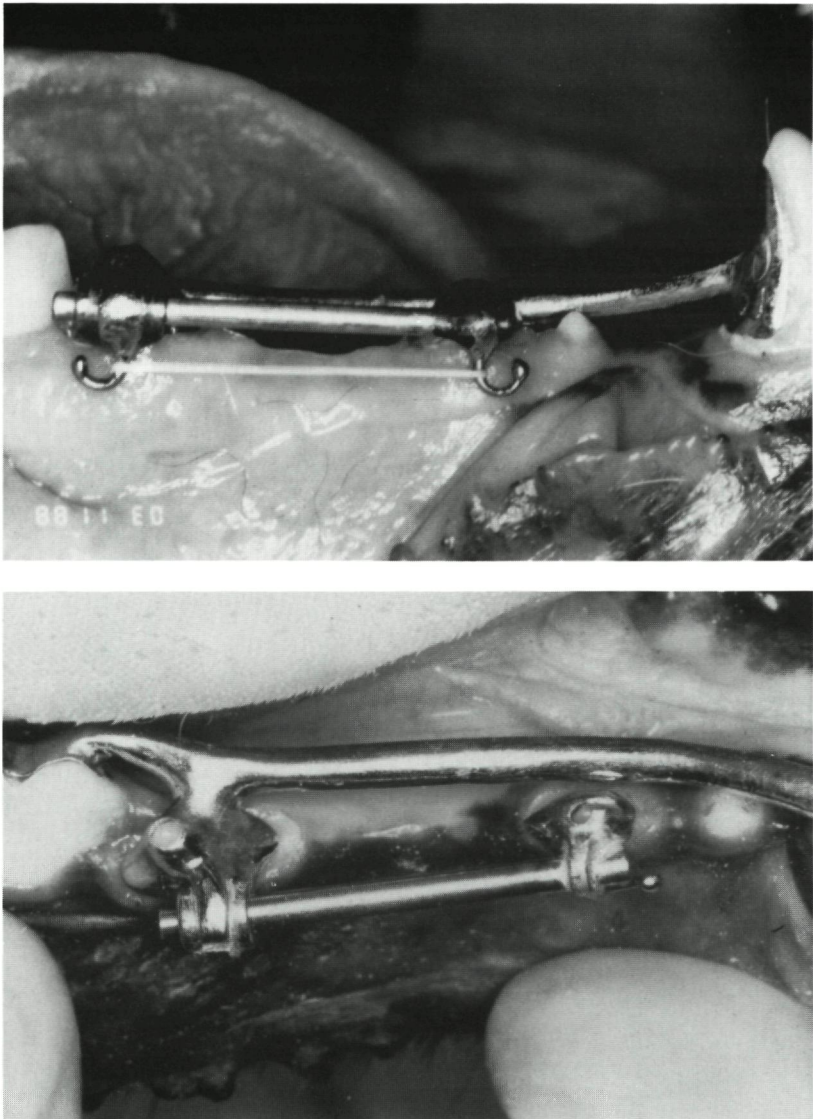


Figure 4-1: *Orthodontic appliance to produce bodily distalization of the second lower premolar. The canine, fourth premolar, and first molar are connected with a lingual bar to serve as an anchorage unit. 1A: buccal view, 1B: occlusal view.*

pre-stretched prior to experimental use. In this way they kept a constant force level for over two weeks¹². Forces were measured twice a week with a strain gauge. New pre-stretched elastics were attached when a deviation of more than 5% of the desired force level was registered. The orthodontic appliance, teeth, and gingiva were thoroughly cleaned twice a week with a toothbrush and gauzes wetted with chlorhexidine 0.02% in water.

4.3.4 Radiographic procedure

Every two weeks standardized oblique lateral radiographs of the left and right side of the lower jaw were taken under general aesthesia. The dogs were fixed in a cephalostat according to Maltha¹³ with two ear rods and a pin in the mid-sagittal plane. The X-ray film (Kodak dental X-ray speed photo, Kodak BV, Driebergen-Rijsenburg, The Netherlands) was placed in a standardized position perpendicular to the central X-ray at a distance of 5 cm behind the side of the mandible. The focus-film distance was 3 m. A Philips Practix X-ray machine (Philips, The Hague, The Netherlands) was used set at 20 mA, and 90 kV with an exposure time of 4 seconds. After exposure films were processed in a R.P.X. Processor (Kodak) for 90 seconds.

4.3.5 Measurements

Twice a week, the distance between the reference points on the orthodontic appliance was measured intraorally with a digital calliper. For each measurement, the dogs were sedated as described before. Each time three successive measurements were made which were averaged for the definitive score.

The radiographs were digitized on an electronic measuring table. The four angular points of the rectangular projection of the buccal tube on the fourth premolar crown and both ends of two of the bone markers were recorded. The centre of the tube was defined as the intersection of the two diagonals. This point was projected on a line connecting the centres of two bone markers (B1 and B2) (Fig. 4-2). The distance between this projection point (Q) and the centre of the mesial bone marker (B1) was used to determine the loss of anchorage in relation to the bone markers.

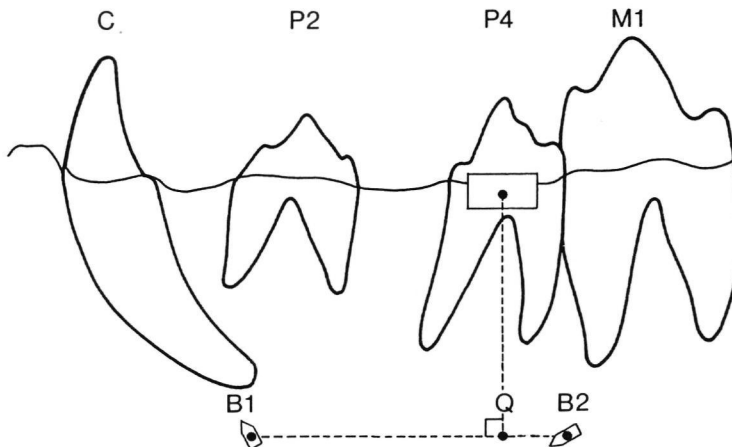


Figure 4-2: *Schematic drawing of the radiographic image. The centre of the tube on the fourth premolar was defined as the intersection of the two diagonals. This point was projected (Q) on the line connecting the centres of two bone markers (B1 and B2). The distance B1-Q was used to determine the loss of anchorage in relation to the bone markers.*

Changes in the distance between the centres of the mesial and distal bone marker (B1-B2) were verified. Every two weeks the dogs were weighed.

4.3.6 Statistics

To assess the reproducibility of the intra-oral measuring method 54 measurements were carried out twice within half an hour by two independent observers. Differences were tested by calculating the standard deviation of the mean difference.

The error of the method for the radiographic study is made up of an error due to inexact positioning of the dog in the cephalostat and a measurement error. In a previous study the error of the radiographic

procedure itself was shown to be negligible¹⁴. Double measurements of 31 radiographs were performed to determine the intra-observer differences. Changes in distance B1-Q and B1-B2 between the force groups were studied with Analysis of Variance (ANOVA).

Time-displacement curves of total tooth movement were plotted for each experimental side. It appeared that these curves could be divided into 4 phases. Two independent observers scored the end of each phase and inter-observer agreement was evaluated. For each phase the duration in days was determined. The relationship between the magnitude of the orthodontic force and the duration of each phase was studied with ANOVA. Pearson correlation coefficients were calculated between mean rates of tooth movement during phase 1, 2, 3, and 4. A combined t-test was performed for each phase to compare the results of the independent samples with the paired samples.

The changes in the distances are the result of the distal movement of the second premolar and the mesial movement of the anchorage unit. Differences in mean loss of anchorage were compared between the different force groups by studying distance B1-Q with ANOVA. The percentage of the total amount of tooth movement during each phase that was caused by mesial movement of the anchorage unit was compared between the force groups with ANOVA and between the phases with the Student's t-test. Pearson correlation coefficients were calculated to study the relationship between the amount of movement of the second premolar and the loss of anchorage. For each phase Pearson correlation coefficients for rate of tooth movement between the left and right side of each dog were calculated. The results of two groups of brother dogs were compared with intra-class correlation coefficients.

The means from first three and the last three weights of the experimental period were compared (Student's t-test).

4.4 Results

4.4.1 General aspects

The measuring technique with the digital calliper was proved to be accurate, as the standard deviation of the mean difference between two observers was 0.02 mm. In the radiographic procedure the error in distance B1-Q was 0.18 mm and intra-observer differences were negligible. The bone markers were proved to be stable as no significant change in distance B1-B2 was found. The weight curves showed a regular increase during the experimental period and a significant weight gain was found.

4.4.2 Tooth movement

Individual time-displacement curves were divided into four phases (Fig. 4-3). Transition from one phase to another in most instances was clear and scores of the two observers were mostly identical. Only for the transition from the acceleration phase (phase 3) to the phase of linear tooth movement (phase 4) differences of one or sometimes two measuring points were found. In these cases a consensus was agreed.

In Table 4-2 the mean duration of phase 1, 2, and 3 is presented.

Table 4-2: *Mean duration (days) and SD of the initial phase (1), phase of arrest of tooth movement (2), and acceleration phase (3) of the second premolar for forces of 50, 100, and 200 cN.*

Phase	50 cN			100 cN			200 cN		
	n	mean	SD	n	mean	SD	n	mean	SD
1	16	4.2	2.0	14	4.0	1.9	14	4.5	1.9
2	16	7.9	9.2	14	7.3	6.7	14	7.1	8.2
3	13	36.6	23.4	11	35.6	21.6	10	38.2	21.5

No significant differences in mean duration of phase 1, 2, and 3 between the force groups (ANOVA, $p \geq 0.1$).

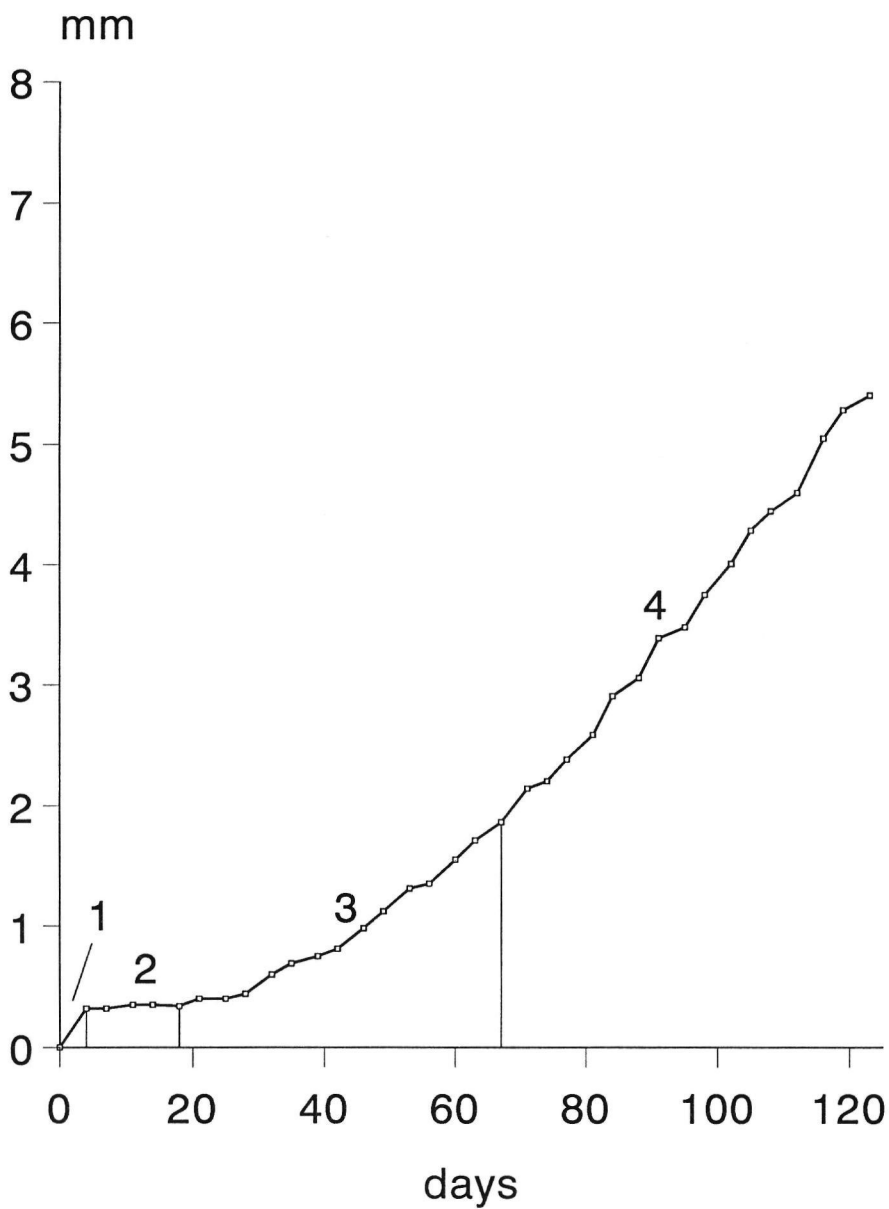


Figure 4-3: *Example of a tooth displacement curve divided into four phases. Phase 1 = initial tooth movement, phase 2 = arrest of tooth movement, phase 3 = acceleration of tooth movement, and phase 4 = constant linear tooth movement.*

In 75% of the cases phase 1 lasted 3 or 4 days or less and its duration was never longer than 7 days. The arrest of tooth movement during phase 2 for all force groups lasted on average about 7 days. In 27% of all cases phase 2 was absent and continuous tooth movement from the beginning to the end of the experiment was observed. In 23% of all cases the acceleration of tooth movement during phase 3 lasted until the end of the experiment; these cases are not listed in Table 4-2. No relationship was found between the duration of phase 1, 2, or 3 and the magnitude of the force (ANOVA, $p \geq 0.1$).

Between the force groups no significant differences in mean rate of tooth movement were found during any phase (ANOVA, $p \geq 0.1$, Table 4-3).

Table 4-3: *Mean rate of tooth movement ($\mu\text{m}/\text{day}$) and SD for the forces of 50, 100, and 200 cN during the initial phase (1), arrest of tooth movement (2), acceleration phase (3), and linear tooth movement (4).*

Phase	50 cN				100 cN				200 cN			
	n	mean	SD		n	mean	SD		n	mean	SD	
1	15	46.4	19.0		13	52.3	14.1		14	62.5	25.9	
2	12	2.8	5.5		10	2.1	13.0		9	0.5	7.5	
3	15	23.6	10.8		13	23.5	9.4		13	22.7	10.8	
4	13	59.3	14.8		11	63.8	19.6		10	62.7	22.1	

No significant differences in mean rate of tooth movement during phase 1, 2, 3, and 4 between the force groups (ANOVA, $p \geq 0.1$).

However, a positive correlation was found between the rate of tooth movement during phase 1 and the magnitude of the force (Pearson 0.33, $p < 0.05$). Mean total tooth movement during phase 1 was 195 μm for 50 cN and 280 μm for 200 cN. The combined t-test showed that no differences exist between the independent and paired samples in each phase. In the control group (0 cN) the mean amount of tooth movement over the whole experimental period was 2 μm .

A large individual variation was found for the mean rate of tooth

movement in all force groups. Differences in individual reaction are illustrated in the time-displacement curves of three experimental sides in different dogs with the same force of 100 cN (Fig. 4-4).

Tooth movement during phase 3 and 4 is continuous, without periods of arrest. Differences in the maximum rate of tooth movement during phase 4 between the force groups are not significant; 72, 76, and 73 $\mu\text{m}/\text{day}$ for 50, 100, and 200 cN respectively. No significant correlation was found between the individual mean rates of tooth movement for the different phases, except for phase 3 and 4 which were highly correlated ($\text{PCC} = 0.69$, $p < 0.01$).

No significant differences in mesial movement of the anchorage unit were found between the three force groups (ANOVA, $p \geq 0.05$). Differences in loss of anchorage as a percentage of the total amount of tooth movement between phase 1, 3, and 4 were not significant. (Student's t-test, $p \geq 0.05$, Table 4-4).

Table 4-4: *Mesial movement of the anchorage unit as a percentage (%) and SD of the total amount of tooth movement for the force groups of 50, 100, and 200 cN during phase 1, 3, and 4.*

Phase	50 cN			100 cN			200 cN		
	n	mean	SD	n	mean	SD	N	mean	SD
1	15	10	38.7	11	2	33.2	13	8	50.5
3	15	38	89.1	11	38	56.4	12	30	45.0
4	13	21	21.6	8	16	19.8	8	21	19.8

No significant differences between the force groups (ANOVA, $p \geq 0.1$) nor between the phases (t-test, $p \geq 0.05$).

The rate of tooth movement during phase 1, 3, and 4 and also the duration of phase 2 were compared between the left and the right side of each dog. A significant correlation between both sides was found for mean rate of tooth movement during phase 3 ($p < 0.05$) and during phase 4 ($p < 0.01$, Table 4-5). Time-displacement curves of the left and right side of each dog

usually are close together, independent of the magnitude of the force (Fig. 4-5). In some cases a marked similarity was found in the time-displacement curves of twin brother dogs (Fig. 4-6). Intra-class correlation coefficients for brother dogs were lower than for the left-right comparison but still significant for the rate of tooth movement during phase 1 ($p < 0.01$) and 4 ($0.01 \leq p < 0.05$) (Table 4-5).

Table 4-5: *Pearson correlation coefficients for mean rate of tooth movement ($\mu\text{m/day}$) during phase 1, 3, and 4 and for mean duration (days) of phase 2 for the left and the right side of each dog. Intra-class correlation coefficients for rate of tooth movement for brother dogs.*

Phase	L-R comparison		Brother-brother comparison	
	n	PCC	n	PCC
1	17	0.22	14	0.59**
2	18	-0.14	8	-0.42
3	17	0.50 *	14	-0.33
4	12	0.92**	9	0.56 *

* $0.01 \leq p < 0.05$

** $p < 0.01$

4.5 Discussion

Since Reitan's experiments^{1,2,4,14,15,16} it is known that the structure of bone has an influence on the rate of orthodontic tooth movement. Although the alveolar bone of dogs is generally denser than in humans¹⁶, differences between the anatomy of the periodontal ligament and alveolar bone of dogs and humans are rather small and in this respect beagles are generally accepted as a good model for comparison with humans¹⁷. In this experiment complete healing of the alveolar bone after extraction of the third premolar was allowed, to provide an experimental site with a uniform bone structure.

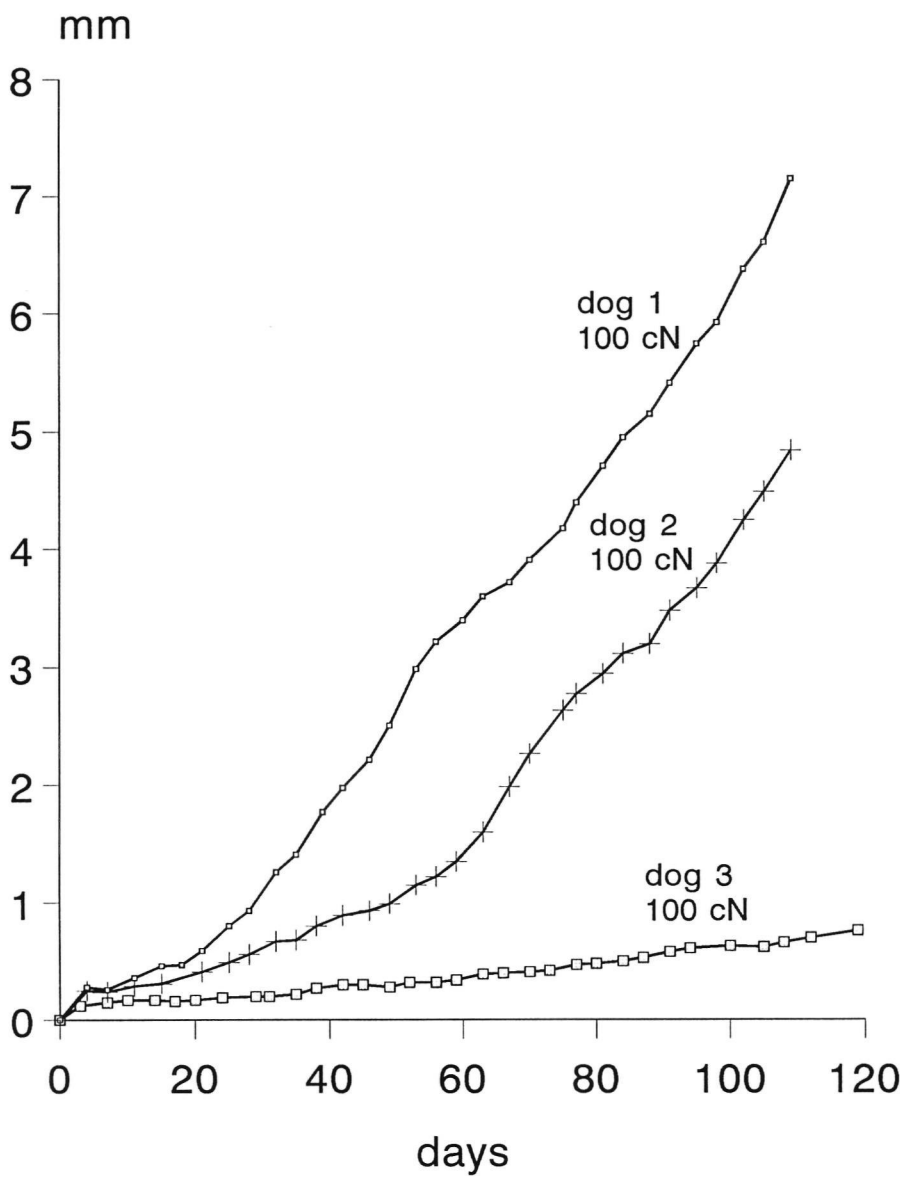


Figure 4-4: *Time-displacement curves of experimental sides of three different dogs with a force of 100 cN.*

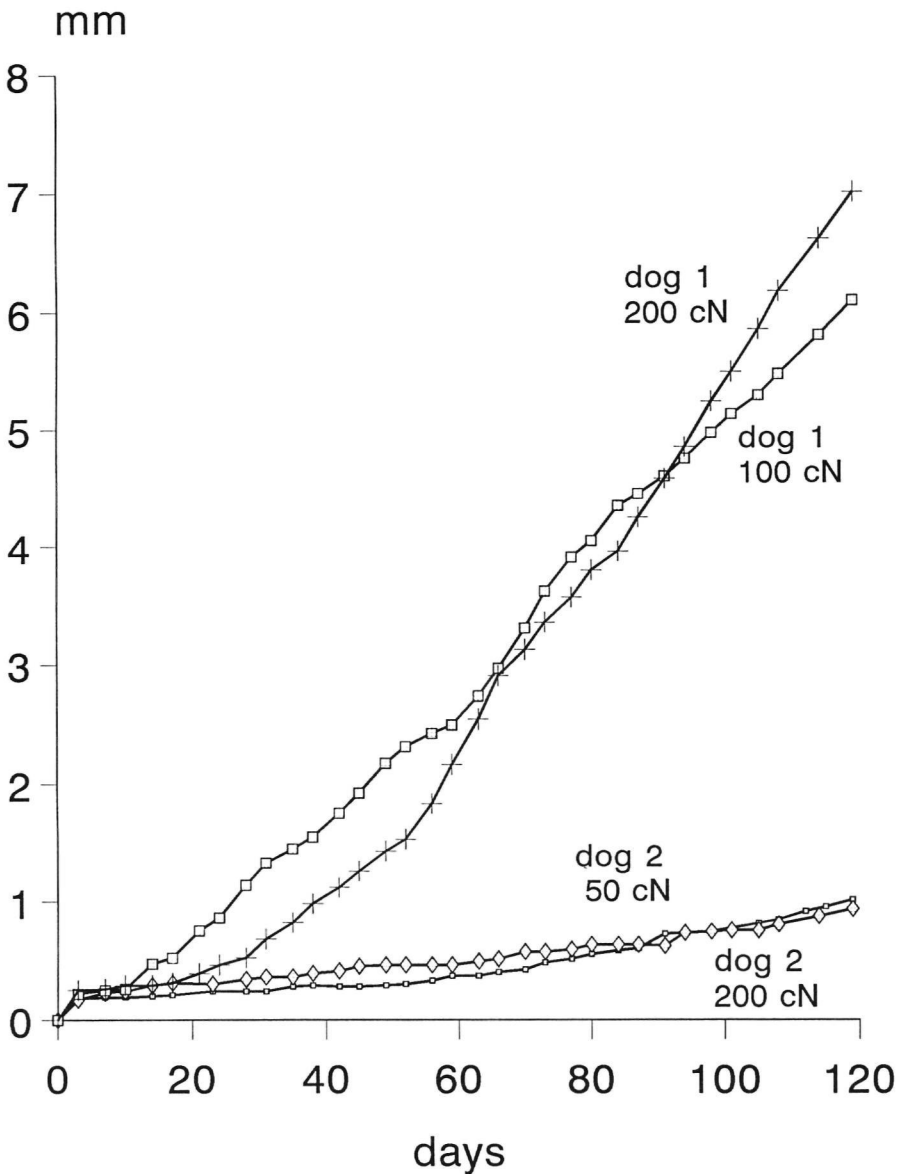


Figure 4-5: Time-displacement curves of the left and the right side of two different dogs. In dog 1 forces of 100 and 200 cN were used, in dog 2 forces of 50 and 200 cN.

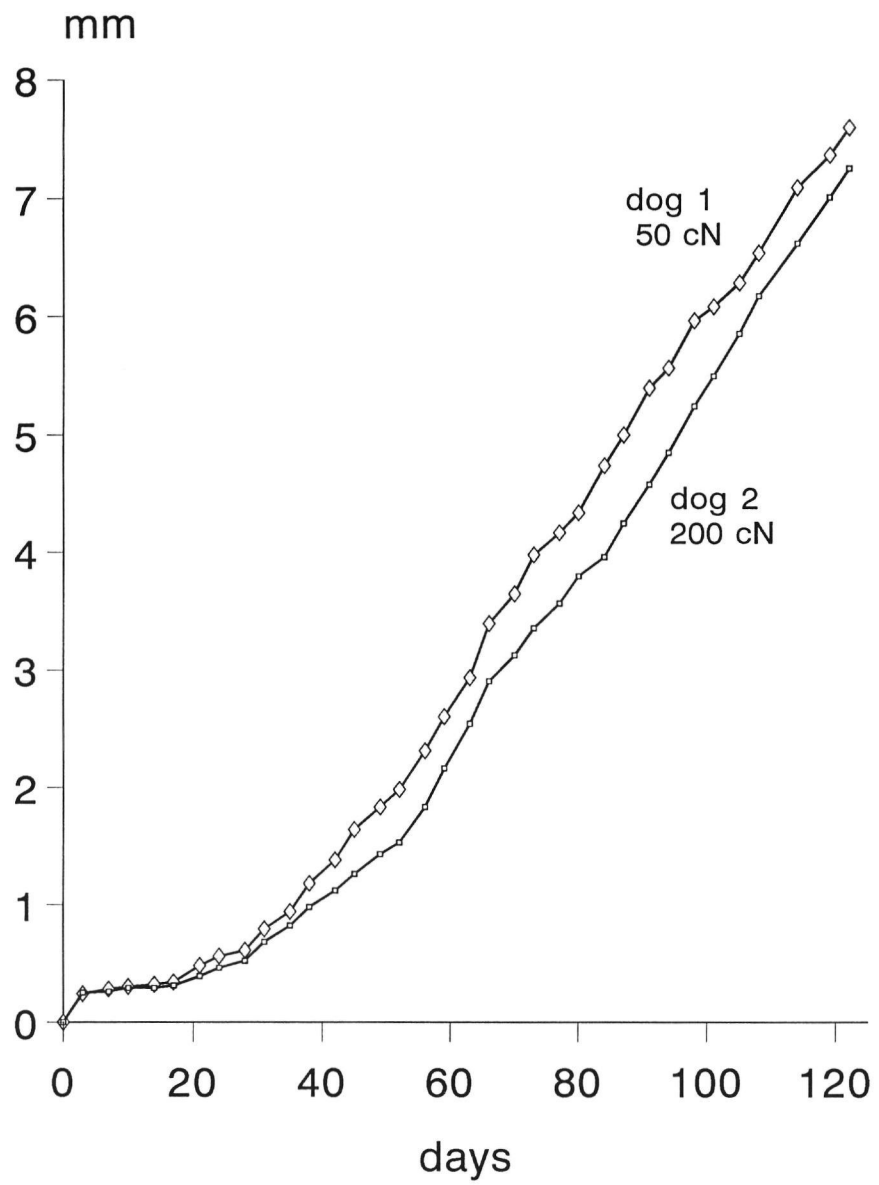


Figure 4-6: *Time-displacement curve of experimental sides of two twin brother dogs with forces of 50 and 200 cN.*

It can be concluded that tooth movement measured in this experiment indeed is the result of the applied orthodontic forces since no significant spontaneous tooth movement was measured in the control group where no elastics were attached. This means that functional forces of tongue or cheek and biting forces do not contribute to the distal movement of the second premolar. Also contraction of transseptal fibres which might occur after extraction of the third premolar, does not produce forces large enough to result in distalization of the second premolar.

The time-displacement curves of the three force groups show a comparable pattern. **Phase 1** can be interpreted as the initial movement of a tooth in its socket. Due to an orthodontic force the width of the periodontal ligament is reduced at the pressure side. This movement is limited by hydrodynamic damping as is described earlier by Bien¹⁸. A great instantaneous movement will be followed by a delayed reaction due to viscoelastic properties of the periodontal ligament. With increasing force the initial tooth movement becomes larger.

Phase 2 can be considered as a period of arrest of tooth movement and is probably associated with hyalinization in the periodontal ligament¹⁵. Hyalinization areas appear earlier and are more extensive if higher forces are used¹⁹. Although the extension of these areas might be related to force magnitude, this seems to have no clinical significance, for bodily tooth movement in the present circumstances, as the duration of phase 2 was independent of the force magnitude. This may be explained by the fact that during bodily tooth movement forces are more equally distributed along the surface of the alveolar bone than during tipping, reducing the risk of hyalinization²⁰. A large individual variation was found in the duration of phase 2, ranging from 0 to 35 days independent of the applied force. This might indicate that differences in bone density or in metabolic activities in bone or periodontal ligament play an important role.

Phase 3 is characterized by a continuous tooth movement with an increasing rate. This acceleration phase may be interpreted as a period in which biologic processes involved in remodelling of the periodontal ligament and alveolar bone reach their maximum capacity. This may also explain why subsequently constant tooth movement is observed in **phase 4**. The small

differences in the maximum rate of tooth movement between the force groups seem to indicate that there is a biological limit for the rate of bodily tooth movement in beagle dogs. Independent of force magnitude, only incidentally no change in tooth position was found between two consecutive measurements during phase 3 and 4. This means that once tooth movement has started, bone remodelling and periodontal ligament turnover takes place at a more or less constant rate.

The root surface area of the distal side of the second lower premolar in beagle dogs is estimated to be 0.5 cm². In the force groups of 50, 100, and 200 cN, initially this would result in a pressure at the root surface of about 10, 20, and 40 kPa respectively. Large differences exist in the optimum pressure advised by different authors: 25-30 kPa²¹, 20-25 kPa²², 7-14 kPa⁶, and 8 kPa²³. In our study however, no significant differences in rate of tooth movement between initial pressures of approximately 10, 20, and 40 kPa could be found. The total root surface area of the anchorage unit is estimated to be 10 times the root surface area of the lower second premolar so initial pressure in the periodontal ligament of these teeth would be about 1, 2 and 4 kPa for the three force groups. Also in the anchorage unit no significant differences in rate of tooth movement were found. So it seems that not only with "high" pressures, but also with "low" pressures in the periodontal ligament the rate of tooth movement is not closely related to force magnitude. This indicates that no linear relationship exists between the initial pressure in the periodontal ligament and the rate of bodily tooth movement.

The actual movement of the second premolar is the difference between the amount of tooth movement measured intra-orally and the mesial movement of the anchorage unit. Calculation of these differences, however, was not justified because of the large difference in the errors between the radiographic and the intra-oral measurements of 0.18 and 0.01 mm respectively.

Large individual differences in the rate of tooth movement were found in all force groups. This is in agreement with previous studies in cats¹. An explanation could be that each individual animal has its own optimum pressure for tooth movement and that in the "slow movers" the optimum forces were not applied. On the other hand, the results of the left and right

side in each dog are highly correlated, although different forces were applied. This might suggest that those "slow movers" were unable to move faster due to lower metabolic capacity resulting in slower bone turn-over. The striking similarity of some time-displacement curves of brother dogs suggests a possible influence of genetic factors. The absence of such similarity in some other twin pairs might be explained by the fact that they were no identical twins.

It must be concluded that other factors than magnitude of force are involved in determining the rate of subsequent tooth movement. Individual differences in bone density, bone metabolism, and turnover in the periodontal ligament may be responsible for the variation. More insight in these factors might lead to the possibility of individual strategies in clinical orthodontic therapy.

4.5 Literature

- 1 REITAN K (1960) Tissue behaviour during orthodontic tooth movement. *Am J Orthod* 46: 881-900.
- 2 REITAN K (1957) Some factors determining the evaluation of forces in orthodontics. *Am J Orthod* 43: 32-45.
- 3 MITCHELL DL, BOONE RM, FERGUSON JH (1973) Correlation of tooth movement with variable forces in the cat. *Angle Orthod* 43: 154-161.
- 4 REITAN K (1967) Clinical and histologic observations on tooth movement during and after orthodontic treatment. *Am J Orthod* 53: 721-745.
- 5 STOREY E, SMITH R (1952) Force in orthodontics and its relation to tooth movement. *Austr J Dent* 56: 11-18.
- 6 BOESTER C, JOHNSTON L (1974) A clinical investigation of the concepts of differential and optimal force in canine retraction. *Angle Orthod* 44: 113-119.
- 7 QUINN R, YOSHIKAWA D (1985) A reassessment of force magnitude in orthodontics. *Am J Orthod* 88: 252-260.
- 8 HIXON EH, AASEN TO, ARANGO J (1969) Optimal force, differential force and anchorage. *Am J Orthod* 55: 437-457.
- 9 BUCK D, CHURCH D (1972) A histologic study of human tooth movement. *Am J Orthod* 62: 507-516.
- 10 BURSTONE C, PRYPYTNIOWICZ R (1980) Holographic determination of

- centres of rotation produced by orthodontic forces. *Am J Orthod* 77: 396-409.
11. BJÖRK A (1968). The use of metallic implants in the study of facial growth in children: method and application. *Am J Phys Anthropol* 29: 243-254.
 12. PILON JJGM, KUIJPERS-JAGTMAN AM, MALTHA JC (1996). Force degradation of orthodontic elastics. *Eur J Orthod* (accepted).
 13. MALTHA JC (1982). The process of tooth eruption in beagle dogs. PhD Thesis, University of Nijmegen, The Netherlands.
 14. WIJDEVELD MGMM, GRUPPING EM, KUIJPERS-JAGTMAN AM, MALTHA JC (1988). Growth of the maxilla after soft tissue palatal surgery at different ages on beagle dogs. *J Oral Maxillofac Surg* 48: 204-209.
 15. REITAN K (1947). Continuous bodily tooth movement and its histological significance. *Acta Odont Scand* 6: 115-144.
 16. REITAN K, KVAM E (1971). Comparative behaviour of human and animal tissue during experimental tooth movement. *Angle Orthod* 41: 1-14.
 17. BARTLEY MH, TAYLOR GN, JEE WS (1970). Teeth and mandible. In: Andersen AC (ed). *The beagle as an experimental dog*. Ames, Iowa: The Iowa State University Press, pp. 189-215.
 18. BIEN S (1966). Hydrodynamic damping of tooth movement. *J Dent Res* 45: 907-914.
 19. RYGH P (1973). Ultrastructural changes in the pressure zones of human periodontium incident to orthodontic tooth movement. *Acta Odont Scand* 31: 109-122.
 20. BURSTONE CJ (1962). The biomechanics of tooth movement. In: Kraus BS, Riedel RA (eds). *Vistas in orthodontics*. Philadelphia (USA): Lea and Febiger, pp. 197-213.
 21. FORTIN JM (1971). Translation of premolars in the dog by controlling the moment-to-force ratio on the crown. *Am J Orthod* 59: 541-551.
 22. JARABAK JR, FIZZELL JA (1963). Technique and treatment with light-wire appliances, light differential forces in clinical orthodontics. St. Louis (USA): CV Mosby Comp., p. 259.
 23. MIURA F, MASAKUNI M, OHURA Y, KARIBE M (1986). The super-elastic property of the Japanese NiTi alloy wire for use in orthodontics. *Am J Orthod Dentofac Orthop* 90: 1-10.

Chapter 5

Orthodontic forces and relapse, an experimental study in beagle dogs

**Jack J.G.M. Pilon
Anne Marie Kuijpers-Jagtman
Jaap C. Maltha**

**American Journal of Orthodontics and Dentofacial Orthopedics, 1996.
In press.**

5.1 Abstract

The relapse after active orthodontic tooth movement was studied. It was related to the magnitude of the experimental orthodontic force and the amount of active tooth movement that was achieved. In 19 young adult male beagle dogs lower third premolars were extracted and bone markers were implanted in the lower jaw. Orthodontic appliances were placed to produce bodily distalization of the lower second premolar, using elastics with a force of 50, 100, and 200 cN respectively. At the left and the right side of each dog different forces were used. After a period of 16 weeks the elastics were removed, while the appliances were left in place. There was no period of retention. Twice a week tooth position was measured directly with a digital calliper from the moment the elastics were removed until tooth movement had arrested for at least 2 weeks. Time-displacement curves of the relapse were plotted. In all cases a period of rapid initial relapse was followed by a gradual decrease in the rate of relapse to a final stable position. The mean amount of relapse was about 40% of the tooth movement that had been produced before, and mean duration of the relapse period was 78 days. No significant differences were found between the force groups, but significant positive correlations were found between the amount of active tooth movement on the one hand and the amount of relapse and the duration of the relapse period on the other hand. Furthermore, significant positive correlations were found between the amount of relapse at the left and the right side, irrespective of the forces that had been applied during active tooth movement.

5.2 Introduction

One of the main problems in orthodontics is relapse after orthodontic tooth movement. The origin of this tendency of teeth to move back to their original position is largely unknown. Several factors have been thought to influence the stabilization of teeth after orthodontic tooth movement. Generally a distinction is made between relapse due to factors within the periodontal ligament and alveolar bone, and other factors such as growth of facial bones, muscular balance, adaptation of soft tissues, and functional aspects¹.

Factors within the periodontal ligament which may be related to relapse are displacement and persistence of the gingival fibre system², persistence of transseptal fibres, especially in extraction sites^{3,4}, recovery of oxytalan fibres^{3,5}, which was later denied by others^{6,7} and the increase in the amount of glycosaminoglycans in the gingiva and changes in their composition^{8,9}. During relapse, periodontal fibres may become rearranged in varying degrees and this may be responsible for differences in the amount of relapse^{10,11}. Changes in structure of and cellular activity in the alveolar bone during orthodontic tooth movement may also influence the process of relapse¹.

Several precautions have been advocated to prevent relapse, such as overcorrection, careful retention after treatment of extremely malposed teeth¹², gingivectomy¹³, and surgical transection of free gingival fibres¹⁴. Before a meaningful discussion can be started about the way relapse can be avoided or diminished, more should be known about relapse without preceding retention. However, a reliable registration of the time-displacement curves of relapse after orthodontic tooth movement has never been made, and the question of the relationship between the force magnitude or the amount of orthodontic tooth movement and the rate and amount of relapse has not yet been answered.

This experiment was started to study the relapse directly after a period in which bodily orthodontic tooth movement was produced by constant forces of three different magnitudes.

5.3 Material and methods

5.3.1 *Experimental set-up*

A group of 19 young adult male beagle dogs was used. The age of the dogs varied between 1 and 1.5 years. Their left and right mandibular third premolars were extracted and three Tantalum bone markers were placed in the inferior border on both sides of the mandible according to the method of Björk¹⁵. Orthodontic appliances were placed at both sides of the lower jaw 16 weeks after the extractions in order to produce bodily distalization of the second premolar. At the left and the right side of each dog different forces were used which were selected at random. Distalization was achieved by the use of elastics producing a force of 50, 100, or 200 cN respectively for 16 weeks. At the end of this period the elastics were removed and the orthodontic appliances were left in place, allowing bodily relapse of the second premolar. The amount of relapse was measured by means of intra-oral measurements and on standardized radiographs.

5.3.2 *Anesthesia*

Extraction of premolars, placement of orthodontic appliances, and radiographic procedures were carried out under general anesthesia. The dogs were premedicated with Thalamonal[®] 1.5 mL (fentanyl 0.05 mg/mL and droperidol 2.5 mg/mL; Janssen Pharmaceutics, Beerse, Belgium) and anesthetized with Nesdonal[®] 15 mg/kg body weight (thiopental sodium 50 mg/mL; Rhone-Poulenc Pharma, Amstelveen, The Netherlands). For intra-oral measurements and oral hygiene measures, the dogs were sedated with 3 mL of a generic preparation containing 10 mg oxycodon HCl, 1 mg acepromazine, and 0.5 mg atropine sulphate per mL which was injected subcutaneous.

5.3.3 *Orthodontic procedure*

Under general anesthesia, extraction of both mandibular third premolars was carried out after hemisection. After 16 weeks, on each mandibular second premolar a crown was made with a rigid sliding bar (ϕ 2.00 mm) soldered at its buccal side, extending distally. A crown was prepared on the permanent

mandibular fourth premolar, and onlays on the permanent canine and first molar. These three teeth were connected with a lingual bar to serve as an anchorage unit. At the buccal side of the crown on the fourth premolar a tube was glued with an inner diameter 0.02 mm larger than the sliding bar of the second premolar. So only bodily distalization of the second premolar was possible by moving the sliding bar through the tube (Fig. 5-1).

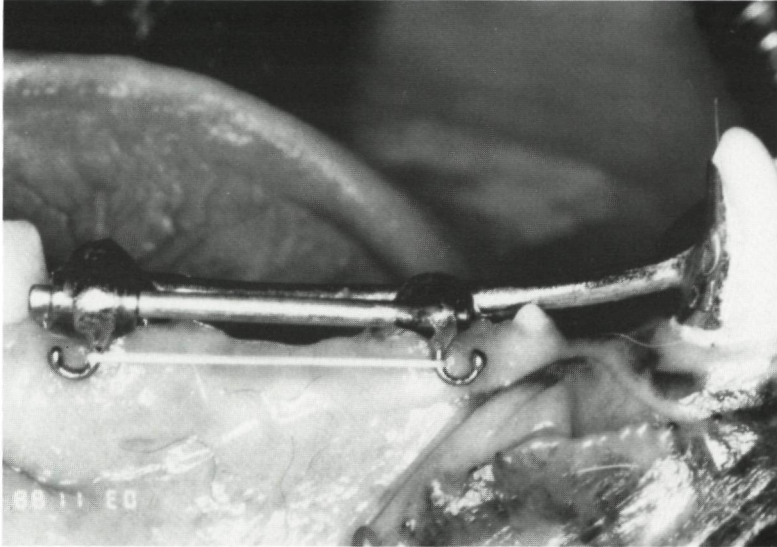


Figure 5-1: *Photograph of the orthodontic appliance with the solid bar attached to a crown on the mandibular second premolar, which slides through a tube on the fourth premolar. A lingual bar connects the canine, fourth premolar and first molar as an anchorage unit.*

The mesial side of the sliding bar and the distal side of the tube on the fourth premolar were prepared parallel to each other to serve as reference planes for the measuring procedure¹⁶. Elastics (Ormco Z-pak elastics, Glendora, Cal., USA) were attached from a buccal hook on the crown of the lower second premolar to a buccal hook on the crown of the lower fourth premolar. The force exerted by each elastic was measured twice a week with a strain gauge. In case a deviation of more than 5% of the desired force level was measured a new elastic was attached. A constant force level for almost three weeks was

obtained by pre-stretching the elastics prior to clinical use¹⁷. The orthodontic appliance and the gingiva were thoroughly cleaned twice a week with a toothbrush and gauzes wetted with chlorhexidine 0.02% in water.

5.3.4 Radiographic procedure

Under general anesthesia, every two weeks lateral radiographs of the left and the right side of the jaws were taken. The dogs were fixed in a cephalostat according to Maltha¹⁸. The distance between the focus and the rotation point of the cephalostat was 3.0 m. Standardized radiographs were taken with a Philips Practix X-ray machine (Philips, The Hague, The Netherlands) which was set at 20 mA and 90 kV with an exposure time of 4 seconds. The films were processed in a R.P.X. processor (Kodak BV, Driebergen-Rijsenburg, The Netherlands) for 90 seconds.

5.3.5 Measurements

After sedation, the distance between the reference points on the orthodontic appliance was measured with a digital calliper. Each time three successive measurements were made, which were averaged for the definitive score. The measurements were performed immediately after removal of the elastics, and further twice a week until relapse had ceased. This was considered to be the case if no change in tooth position was found for 2 weeks.

The radiographs were digitized on an electronic measuring table. The four angular points of the rectangular projection of the buccal tube on the fourth premolar crown and both ends of two of the bone markers were recorded. The centres of the bone markers were defined as the midpoints of the axial lines connecting the two ends of each marker. The connecting line between those centres was denoted B1-B2 (Fig. 5-2). The centre of the tube was defined as the intersection of its two diagonals. This point was projected on line B1-B2, resulting in point Q. The distance Q-B1 was used to determine the movement of the anchorage unit in relation to the bone markers. The stability of the bone markers themselves was verified by changes in the distance between the centres of both bone markers (distance B1-B2).

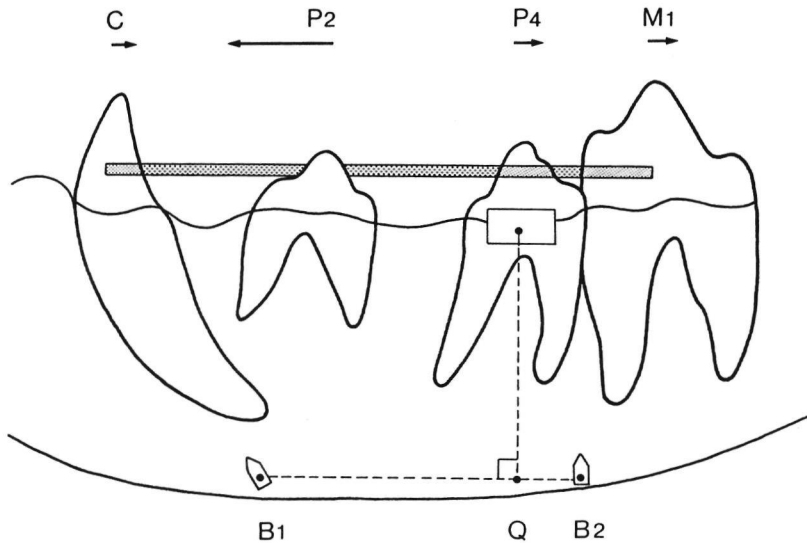


Figure 5-2: *Schematic representation of the canine (C), second premolar (P2), fourth premolar (P4), and first molar (M1) on lateral radiographs. The centre of the tube on the fourth premolar was projected on the line connecting the centre of the bone markers B1 and B2, resulting in point Q. The dotted bar represents the anchorage unit of C, P4, and M1, arrows indicate direction of relapse.*

5.3.6 Statistics

Time-displacement curves of tooth movement during relapse were plotted for each experimental side. Two independent observers scored the end of the relapse period, and the inter-observer agreement was evaluated. The total amount of active tooth movement, the total amount of relapse, and the proportional relapse were calculated. Proportional relapse was defined as the amount of relapse as a percentage of the total amount of active tooth movement. These values were compared between the force groups with ANOVA. The time-displacement curves were studied by comparing duration, the position of the half-way point, and mean rate of relapse between the force groups with ANOVA. The halfway point (T1/2) of the relapse curve was defined as the moment at which half of the total amount of relapse was

completed. Because the left and the right side of each dog are incorporated in different force groups, the results are not independent. Therefore a combined t-test was performed to compare the results of the paired with the independent samples. The percentage of the total relapse, caused by the distal movement of the anchorage unit, was calculated for the different force groups and compared with ANOVA.

Pearson Correlation Coefficients (PCC) were calculated between the total amount of tooth movement during the active treatment on the one hand, and the amount of relapse, the proportional relapse, and the duration of the relapse period on the other hand. For comparison between the right and left side, PCC's were calculated for the amount of relapse, the halfway point, and the proportional relapse.

5.4 Results

During the experimental period weight curves of the dogs showed a regular increase and no health problems occurred. The bone markers proved to be stable, as no changes in distance B1-B2 were found. In a previous study¹⁶, the error in the measuring procedure with the digital calliper was shown to be small: the SD of the mean difference between two independent observers was 0.02 mm.

An example of a time-displacement curve during relapse is shown in Figure 5-3. A period of rapid initial relapse is followed by a gradual decrease in the rate of relapse until a stable position is reached. The determination of the end of the relapse by the two observers was identical in nearly all cases. Only in a few cases differences of one or two measuring points were found and in these cases a consensus was agreed.

None of the parameters calculated, mean amount of tooth movement during active distalization, mean amount of relapse, mean proportional relapse, mean duration, mean position of the halfway point, and mean rate of relapse, showed significant differences between the force groups (ANOVA, $p > 0.1$, Table 5-1). The combined t-test showed that no differences exist between the paired and the independent samples.

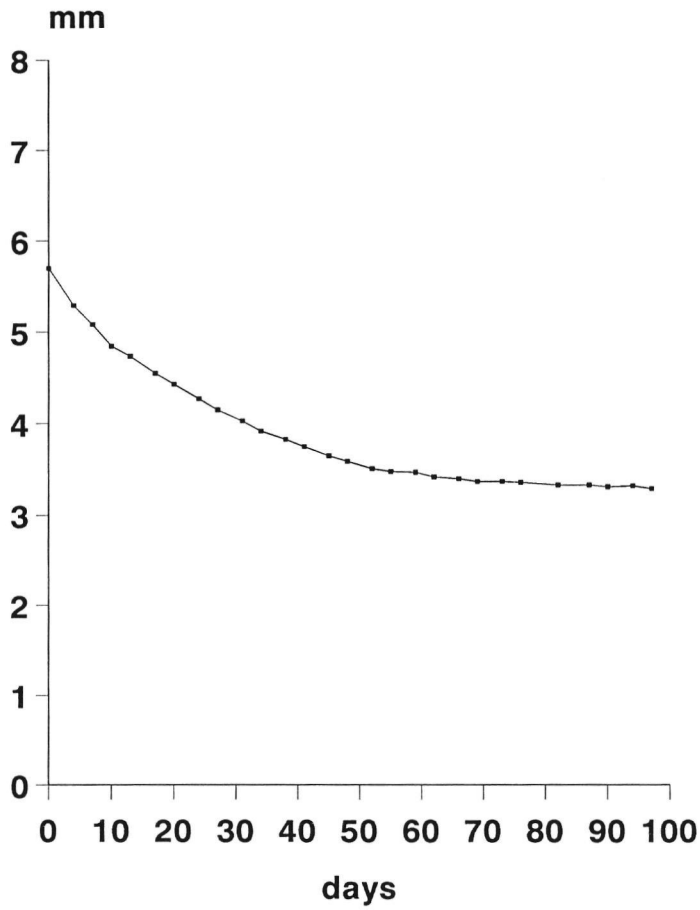


Figure 5-3: *An example of a time-displacement curve during relapse.*

A large individual variation in duration of the relapse period was found in all force groups (range 45-108 days). The time-displacement curves of the relapse at the experimental sides of three different dogs with the same amount of active tooth movement, illustrate the individual variation that exists in the amount of relapse (Fig. 5-4). Still, significant correlations were found between the amount of active tooth movement and the amount of relapse afterwards ($PCC = 0.86, p < 0.01$), and also for the amount of active tooth movement and the duration of the relapse period ($PCC = 0.62, p < 0.01$).

Table 5-1: *Mean amount of tooth movement with orthodontic forces of 50, 100, and 200 cN during 16 weeks, mean amount of relapse, mean proportional relapse, mean duration of the relapse period, mean position of the halfway point T1/2, and mean rate of tooth movement during relapse. n = number of experimental sides.*

Force n	50		100		200	
	14		12		12	
	mean	SEM	mean	SEM	mean	SEM
Tooth movement (mm)	4.7	0.4	4.1	0.6	4.2	0.7
Relapse (mm)	1.9	0.2	1.5	0.2	1.8	0.3
Relapse (%)	39		37		42	
Duration (days)	76.8	3.7	75.2	4.8	81.7	5.6
T1/2 (days)	21.0	2.0	21.3	4.8	20.7	2.3
Relapse rate ($\mu\text{m}/\text{day}$)	24.1	1.7	19.9	2.8	21.0	2.8

No significant differences between the three force groups (ANOVA, $p > 0.1$).

If the left and right side of each dog were compared, a significant correlation was found for the amount of relapse ($\text{PCC} = 0.70$, $p < 0.01$) as well as for the halfway point ($\text{PCC} = 0.64$, $p < 0.01$). A weak positive correlation was found between the proportional relapse on the right and left side ($\text{PCC} = 0.38$, $0.05 \leq p < 0.1$).

The changes in distance that were measured intra-orally are the resultant of the mesial relapse of the second premolar and distal relapse of the anchorage unit. The measurements on the radiographs revealed a mean distal movement of the anchorage unit of $0.9 \mu\text{m}/\text{day}$. The percentage of the total amount of relapse caused by the distal movement of the anchorage unit showed no significant differences between the force groups (ANOVA, $p > 0.1$).

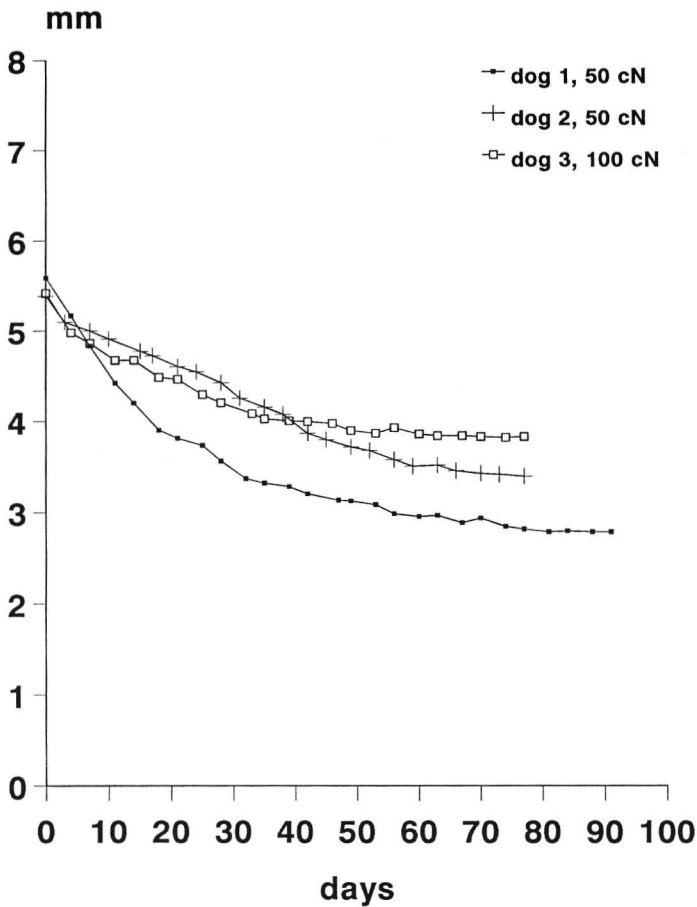


Figure 5-4: *Time-displacement curves of the relapse of three different dogs with the same amount of active tooth movement of 5.5 mm.*

5.5 Discussion

The alveolar bone in beagle dogs is generally denser than in humans¹⁹, which has an influence on rate of orthodontic tooth movement²⁰. Still, differences between the anatomy of the alveolar bone and the periodontal ligament of dogs and humans are so small that dogs are generally accepted as a suitable experimental model for comparison with humans²¹.

In a previous experiment, in which similar orthodontic appliances were placed without elastics, it was shown that tooth movement was not caused by functional forces from tongue or cheek, biting forces, or growth of the jaws¹⁶. This means that in the present study relapse can only be attributed to forces originating from the periodontal ligament and the alveolar bone. The appliances were left in place during relapse in order to prevent tipping movements.

The time-displacement curves during bodily relapse show a similar course in all cases. The fact that no significant differences in the halfway point were found stresses this uniformity. However, some extreme individual values were found in the amount and duration of relapse. The same kind of individual differences were also found in the time-displacement curves during active tooth movement¹⁶. In earlier research, Reitan²⁰ has discussed the individual variation in the structure of and cellular activity in the periodontal ligament and alveolar bone. In his opinion this might be associated with individual differences in tooth movement characteristics. The results of the present study are in agreement with his point of view, as the total amount of relapse as well as the half-way point show significant correlation between the left and the right side of each dog. A previous study has already revealed that also the amount of active tooth movement at the left and right side of a dog are highly correlated, irrespective of the magnitude of the orthodontic forces¹⁶. So, individual characteristics seem to determine the rate of both active tooth movement and relapse.

The rate of relapse of the anchorage unit was about 5% of the total relapse. In a former study¹⁶, it was shown that there was no movement of the anchorage unit in dogs of a control group, where the appliances were not activated. In the same study, the total error of the measuring procedure on the radiographs appeared to be 0.18 mm, which is about ten times larger than the error in the intra-oral measurements. Therefore the results of both measurements could not be combined to calculate the actual mesial relapse of the second premolar. The root surface area of the anchorage unit of canine, fourth premolar and first molar was estimated to be 10 times that of the second premolar. Theoretically, during tooth movement the pressure in the periodontal ligament of the anchorage unit would then be 10 times as low as

that of the second premolar. No differences in relapse of the anchorage unit were found between the force groups. So it seems that also with these very low pressures in the periodontal ligament, the magnitude of an orthodontic force is not decisive for the rate of tooth movement.

It is known that the periodontal ligament shows visco-elastic properties and the recovery after the application of a force of short duration is time dependent^{22,23}. A schematic representation of this type of tooth movement is given by the exponential stress-strain curve of a Voigt element. In this element a spring and dashpot are in parallel. This model has also successfully been used in the description of characteristics of other tissues in which fibrous material is internally damped by liquid in the interstices^{23,24}. From the present experiment it becomes clear that also during relapse, the curve representing the long term recovery of the tooth surrounding structures, suggests visco-elastic properties. However, the situation in this case is far more complicated, as not only an immediate reaction of fibres and interstitial fluids takes place, but also a long term reorganization of the periodontal ligament and alveolar bone, involving complex cellular activities.

The initial behaviour of a tooth after release of orthodontic forces, suggests that an opposite force is generated by the surrounding tissues. Its origin is still unclear, but in one way or another it seems to be related to the amount of tooth movement during active treatment. This suggests that energy or information is stored in the periodontal ligament and alveolar bone during orthodontic tooth movement. The mechanism to absorb this energy is not yet understood. Elongation and compression of transseptal fibres at the tension- and pressure-side of the socket may be involved¹². The length and direction of supracrestal collagenous fibres are known to adjust extremely slow to orthodontic tooth movement⁵, although on the other hand high turnover rates have been reported²⁵. Recoverable changes in the physical state of the collagenous matrix of the periodontal ligament, which is considered to have properties of a thixotropic gel²⁶, may be important. According to Bien²⁷ also the vascular system is involved in fluid transmission, distribution, and dissipation of kinetic energy in the periodontal ligament. The recovery of the compressed blood vessel system may contribute to increased tissue pressure at the former compression side in the periodontal ligament when orthodontic

forces are removed.

In a previous study it was shown that the maximum rate of active tooth movement by forces of 50, 100, or 200 cN was approximately $75 \mu\text{m/day}$ ¹⁶. The mean rate of tooth movement during the total relapse period is $22 \mu\text{m/day}$. During the rapid initial phase of relapse however, the rate of tooth movement can be as high as $100 \mu\text{m/day}$ over a period of 2 weeks. Apparently the biological limit for the rate of bodily tooth movement exceeds the maximum rate of tooth movement of $75 \mu\text{m/day}$, as found in previous experiments¹⁶. One could imagine that this high rate of tooth movement is allowed by a widened periodontal space at the former tension side. A histological study²⁸ showed indeed that the width of the periodontal space had increased to 200-250 μm , but this is far too narrow to explain the present findings. It is more likely that the newly formed bundle bone on the former tension side is more easily resorbed than mature alveolar bone, as was already suggested by Reitan¹².

The fact that relapse starts immediately after removal of the orthodontic force indicates that during orthodontic tooth movement the effective force is in fact the resultant of an active orthodontic force and a reactive opposite force by the surrounding tissues. As soon as the orthodontic force is removed the resulting reactive forces initiate immediate relapse within the socket. After this initial reaction, cellular response leads to differentiation of osteoclasts at the former tension side^{11,12,28}.

As the amount and rate of relapse is strongly correlated with the amount of the preceding active movement, it is likely that information of some kind is stored in the biological system during that period. The nature of this information is not yet known, but it might be stored in a physical or chemical way. To induce a prolonged tissue response, as is the case during relapse, one has to assume that this information is released and slowly disappears in the course of the relapse period.

To diminish or prevent relapse in the clinical situation, the question is which measures are most effective. A proper discussion on these measures requires more knowledge on the nature of its cause than is available at this moment.

5.6 Literature

1. REITAN K (1969). Principles of retention and avoidance of posttreatment relapse. *Am J Orthod* 55: 776-790.
2. ATHERTON JD (1970). The gingival response to orthodontic tooth movement. *Am J Orthod* 58: 179-186.
3. ERIKSON BE, KAPLAN H, AISENBERG MS (1945). Orthodontics and transseptal fibres. *Am J Orthod* 31: 1-20.
4. PARKER GR (1972). Transseptal fibres and relapse following bodily retraction of teeth: A histologic study. *Am J Orthod* 61: 331-344.
5. EDWARDS JG (1968). A study of the periodontium during orthodontic rotation of teeth. *Am J Orthod* 54: 441-461.
6. SIMS MR (1976). Reconstruction of the human oxytalan system during orthodontic tooth movement. *Am J Orthod* 70: 38-58.
7. BOWLING K, RYGH P (1988). A quantitative study of oxytalan fibres in the transseptal region and tension zones of rat molars following orthodontic tooth movement. *Eur J Orthod* 10: 13-26.
8. RÖNNERMAN A, THILANDER B, HEYDEN G (1980). Gingival tissue reaction to orthodontic closure of extraction sites. *Am J Orthod* 77: 620-625.
9. KUROL J, RÖNNERMAN A, HEYDEN G (1982). Long-term gingival conditions after orthodontic closure of extraction sites. Histological and histochemical studies. *Eur J Orthod* 4: 87-89.
10. REITAN K (1959). Tissue rearrangement during retention of orthodontically rotated teeth. *Angle Orthod* 29: 105-113.
11. REITAN K (1967). Clinical and histologic observations on tooth movement during and after orthodontic treatment. *Am J Orthod* 53: 721-745.
12. REITAN K (1960). Tissue behaviour during orthodontic tooth movement. *Am J Orthod* 46: 881-899.
13. BOESE LR (1969). Increased stability of orthodontically rotated teeth following gingivectomy in *Macaca nemestrina*. *Am J Orthod* 56: 273-290.
14. BRAIN WE (1969). The effect of surgical transection of free gingival fibres on the regression of orthodontically rotated teeth in the dog. *Am J Orthod* 55: 50-70.
15. BJÖRK A (1968). The use of metallic implants in the study of facial growth in children: method and application. *Am J Phys Anthropol* 29: 243-254.

16. PILON JJGM, KUIJPERS-JAGTMAN AM, MALTHA JC (1996). Magnitude of orthodontic forces and rate of bodily tooth movement - an experimental study in the beagle dog. *Am J Orthod Dentofac Orthop* 110: 16-23.
17. PILON JJGM, KUIJPERS-JAGTMAN AM, MALTHA JC (1996). Force degradation of orthodontic elastics. *Eur J Orthod* (accepted).
18. MALTHA JC (1982). The process of tooth eruption in beagle dogs. PhD Thesis, University of Nijmegen, The Netherlands.
19. REITAN K, KVAM E (1971). Comparative behaviour of human and animal tissue during experimental tooth movement. *Angle Orthod* 41: 1-14.
20. REITAN K (1957). Some factors determining the evaluation of forces in orthodontics. *Am J Orthod* 43: 32-45.
21. BARTLEY MH, TAYLOR GN, JEE WS (1970). Teeth and mandible. In: Andersen AC (ed). *The beagle as an experimental dog*. Ames, Iowa: The Iowa State University Press, pp. 189-215.
22. KARDOS TB, SIMPSON LO (1979). A theoretical consideration of the periodontal membrane as a collagenous thixotropic system and its relationship to tooth eruption. *J Period Res* 14: 444-451.
23. PICTON DC, WILLS DJ (1978). Viscoelastic properties of the periodontal ligament and mucous membrane. *J Prosthet Dent* 40: 263-272.
24. WILLS DJ, PICTON DC, DAVIES WI (1972). An investigation of the viscoelastic properties of the periodontium in monkeys. *J Periodont Res* 7: 42-51.
25. DEPORTER DA, SVOBODA ELA, HOWLEY TP, SHIGA A (1984). A quantitative comparison of collagen phagocytosis in periodontal ligament and transseptal ligament of the rat periodontium. *Am J Orthod* 85: 519-522.
26. KARDOS TB, SIMPSON LO (1980). A new periodontal membrane biology based upon thixotropic concepts. *Am J Orthod* 77: 508-515.
27. BIEN SM (1966). Hydrodynamic damping of tooth movement. *J Dent Res* 45: 907-914.
28. PILON JJGM, MALTHA JC, KUIJPERS-JAGTMAN AM (1996). Histology of the periodontal ligament during bodily orthodontic tooth movement in beagle dogs. *Eur J Orthod* (submitted).

Chapter 6

Histology of periodontal ligament and alveolar bone during bodily orthodontic tooth movement in beagle dogs

Jack J.G.M. Pilon
Jaap C. Maltha
Anne Marie Kuijpers-Jagtman

Submitted to the European Journal of Orthodontics, 1995.

6.1 Abstract

Histologic changes in the periodontal ligament during orthodontic tooth movement were studied in beagle dogs. Mandibular second premolars were bodily moved with constant forces of 50, 100, and 200 cN. Histologic evaluation was carried out during active tooth movement and during relapse. At the pressure side of the periodontal ligament normal tissue structure was initially lost, followed by undermining resorption in hyalinized areas. Later, fibres were oriented parallel to the root surface and direct osteoclastic activity was strictly limited to local bony protrusions. The resorptive activity differed between the bone and root related part of the periodontal ligament. At the tension side the gross fibrous structure was initially undisturbed, and osteoblasts differentiated within seven days. The rate of bone deposition increased and trabecular bone was formed. The width of the periodontal ligament was increased and cellular cementum was deposited along the root surface. At the tension side less damage in the periodontal ligament was observed than at the pressure side. Apart from small differences in initial reaction, histologic changes in the periodontal ligament were independent of force magnitude. Root resorption seemed to increase with the amount and duration of tooth movement. During relapse the patterns of bone resorption and deposition had reversed, and root resorption was present at the former tension side.

6.2 Introduction

Tooth movement is a physiologic process, which acts as an adaptive mechanism to balance the effect of orthodontic or natural forces on teeth by cellular and tissue responses within the periodontal ligament. Histological changes in the periodontal ligament of orthodontically moved teeth were first reported by Sandstedt (1904). Resorption of bone was found at the "pressure" side and deposition at the "tension" side. According to Schwartz (1932), damage to the periodontal ligament could be avoided if an orthodontic force was kept smaller than the capillary blood pressure, or when its range of activation would be less than the width of the periodontal ligament (Stuteville, 1938). To minimize the risk of pathologic reactions, as hyalinization, Reitan (1960) advised to avoid stress concentrations in the periodontal ligament by the use of low forces and the avoidance of tipping tooth movements. Burstone (1962) showed that in bodily tooth movement the force is evenly distributed along the surface of bone and root, thus reducing the risk of hyalinization and subsequent arrest of tooth movement.

Storey (1973) distinguished three possible reactions of periodontal tissues to forces. First, a bio-elastic reaction to forces of short duration, allowing reversible movement of the tooth within its socket. Secondly, bio-plastic behaviour results in adaptive tissue changes allowing tooth movement, and thirdly, a bio-disruptive deformation is found in case forces are too high. The effectiveness of orthodontic therapy thus depends on the mechanical circumstances in the periodontal ligament, and Burstone (1989) stressed the importance of an optimal force system for an adequate biological response in the periodontal ligament.

Series of direct intra-oral measurements revealed that the time-displacement curve of active bodily orthodontic tooth movement can be divided into four phases (Pilon *et al.*, 1996; Fig. 6-1). In the first phase, the tooth probably moves in its socket. Histological changes in the periodontal ligament during this phase have been described extensively (Rygh, 1972; King and Fischlschweiger, 1982; Kuitert, 1988). This initial response seems to be the result of vascular, neural and bio-electrical reactions (Davidovitch *et al.*, 1989).

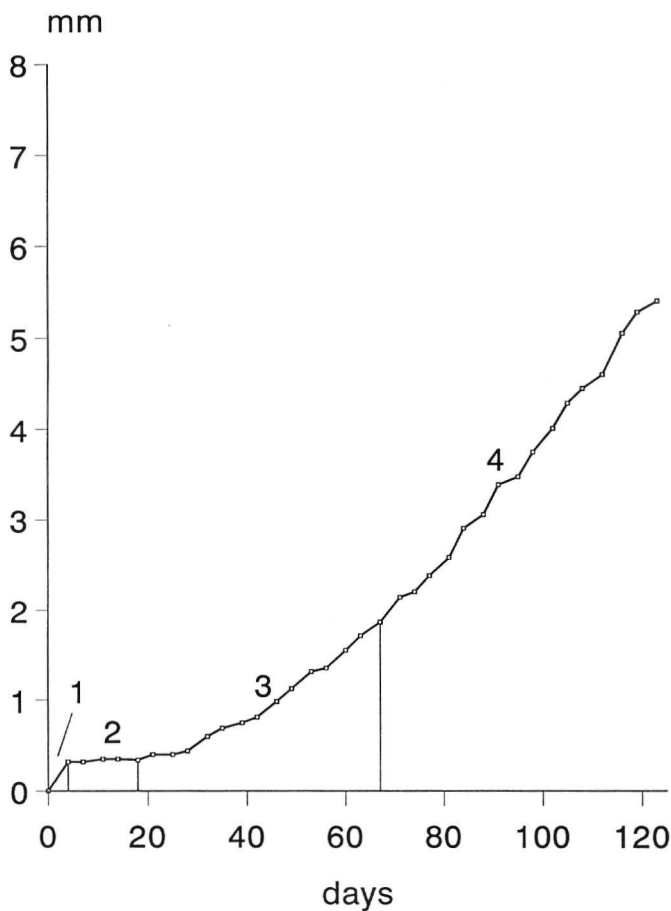


Figure 6-1: *Individual time-displacement curve used for the determination of the different phases of orthodontic tooth movement.*

Increased pressure in the periodontal ligament induced a diminished vascular supply (Rygh *et al.*, 1986). Hyalinization in the periodontal ligament is associated with arrest of tooth movement in the next phase. Hyalinized zones can be expected in all clinical orthodontic treatment (Reitan, 1969). The degenerative changes increased with time in cases of continuous pressure and larger hyalinization zones were found with higher force levels (Reitan, 1964).

Removal of the necrotic or hyalinized tissue and bone resorption from the adjacent marrow spaces were already described as "undermining resorption" by Sandstedt (1904).

The third phase of the time-displacement curve is characterized by an increase in the rate of tooth movement. During the next phase the rate of tooth movement finally reaches a constant level (Pilon *et al.*, 1996). The complex process of osteoclastic bone resorption and the histochemical reactions involved during orthodontic tooth movement, were described by several authors (e.g. Davidovitch, 1979; Yamasaki, 1989). However, very little is known about the histologic changes in the periodontal ligament during these last two phases, during which by far most of the orthodontic tooth movement takes place, and about the dependence of these histologic changes on force magnitude. The same holds true for tissue changes during relapse.

Therefore a study was performed in order to describe the histologic changes in the periodontal ligament of beagle dogs during bodily orthodontic tooth movement using constant and continuous forces of different magnitude and during the subsequent relapse.

6.3 Materials and methods

6.3.1 Experimental set-up

Eight young adult male beagle dogs were used for the present study. The age of the dogs at the start of the study varied between 1 and 1.5 years. After premedication with 1.5 mL Thalamonal^R (Fentanyl 0.05 mg/mL and Droperidol 2.5 mg/mL; Janssen Pharmaceutica, Beerse, Belgium) and subsequent general anaesthesia with 15 mg/kg Nesdonal^R (Thiopental Sodium 50 mg/mL; Rhone-Poulenc Pharma, Amstelveen, The Netherlands), mandibular third premolars were extracted after hemisection. Sixteen weeks later orthodontic appliances were placed on the left and right sides of each dog. The second premolars were bodily distalized with forces of 50, 100, or 200 cN, as described elsewhere (Pilon *et al.*, 1996). Bodily relapse of the second premolar was allowed immediately after removal of the orthodontic force. Time-displacement curves of the active orthodontic tooth movement

and the relapse were plotted and compared with the curves obtained from a previous study (Pilon *et al.*, 1996; Fig. 6-1) in order to determine a time schedule for histological evaluation.

The dogs were killed at selected moments of their time-displacement curves and both sides of the mandible were prepared for histologic evaluation (Table 6-1).

Table 6-1: *Division of the experimental sides over the different phases. Forces F (cN) and the duration D (days) of the force application. Duration R (days) of relapse.*

Phase 1		Phase 2		Phase 3 and 4		Relapse	
F	D	F	D	F	D	F	R
50	7	50	14	50	81	50	18
100	7	100	14	100	81	50	91
200	7	200	14	200	119	100	109
		200	21				
		200	25				

For phase 1 three sides were used to which forces had been applied of 50, 100, and 200 cN respectively for a period of 7 days. To study phase 2, three sides were studied 14 days after the application of forces of 50, 100, and 200 cN respectively, one side after 21 days with 200 cN, and one side after 25 days with 200 cN. For phases 3 and 4 one side was used after 81 days application of a force of 50 cN and another one with 100 cN, and one side was used after 119 days with 200 cN. During relapse one side was studied 18 days after removal of the elastics, and two sides at the end of the relapse, when the teeth had come to a standstill. One dog, which had no treatment at all, was used as a control.

6.3.2 Histological procedures

The dogs were anaesthetized with Narcovet^R (sodium pentobarbital 60 mg/mL, Apharmo, Arnhem, The Netherlands). Then 0.5 mg/kg heparin

(Thromboliquine^R, Organon, Boxtel, The Netherlands) was given, followed by a lethal dose of Narcovet^R intravenously after some minutes. The thorax was opened and the vascular system was perfused by the aortic arch with physiological saline, followed by 4% neutral formaldehyde for fixation. Both sides of the mandible were dissected and immersed in a 4% neutral formaldehyde solution for 2 weeks. They were sawed in smaller blocks, demineralized in 20% formic acid and 5% sodium citrate, dehydrated, and embedded in Paraplast^R (Monoject Scientific Inc., Athy, Ireland). Serial mesio-distal sections of 7 μm were cut and stained with Haematoxylin and Eosin or a modification of Herovici's pentachrome staining which is indicative for type I and type III collagen (Levame and Meyer, 1987).

6.4 Results

6.4.1 *Active tooth movement, pressure side*

In phase 1, seven days after the application of an orthodontic force, direct bone resorption was observed especially in the middle third part of the periodontal ligament. There were no cell-free zones, but the course and attachment of the collagenous fibres was disturbed. In the vicinity of osteoclasts Sharpey's fibres were absent. The number of blood vessels was normal and they were only slightly compressed. In the case of 50 cN the width of the periodontal ligament was more or less uniform and amounted 100-200 μm . Osteoclastic activity was most prominent with a force of 200 cN (Fig. 6-2). In this case, the bone surface was very irregular due to active localized resorption areas, and the width of the periodontal ligament varied between 25-400 μm . In no case direct contact between root and alveolar bone was found. Resorptive activity was much higher at the bone than at the root surface and the attachment was far more lost at the bone side. In all cases, small spots with local resorption were observed at the root surface, most prominent with a force of 200 cN. There also the most severe distortion of the fibre arrangement was seen.

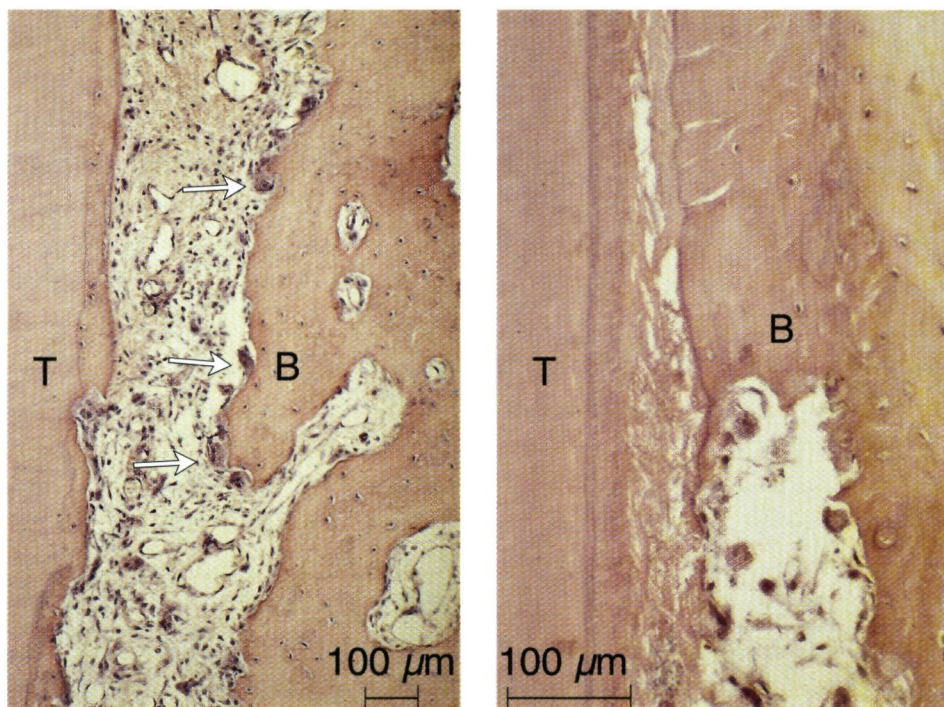


Figure 6-2: *Photomicrograph showing the periodontal ligament at the pressure side of a second premolar to which a force of 200 cN was applied for 7 days.*

T = Tooth; B = Alveolar bone; arrows indicate osteoclasts.

Haematoxylin and Eosin; neg magn x 25.

Figure 6-3: *Photomicrograph showing the periodontal ligament at the pressure side of a second premolar to which a force of 200 cN was applied for 14 days. Hyalinization of the periodontal ligament and localized undermining resorption are apparent.*

T = Tooth; B = Alveolar bone.

Haematoxylin and Eosin; neg magn x 63.

In phase 2, after 14 days, cell-free areas were present in all cases in the middle third part of the periodontal ligament. Direct resorption next to the borders of the hyalinization area was accompanied by undermining resorption in the most narrow part of the periodontal ligament (Fig. 6-3), which had an average width of about 75 μm . The course of the collagenous fibres was completely disturbed and the periodontal ligament showed severe compression in the hyalinized areas. In other areas no deviations in the fibre arrangement were present. Large differences were found in local cellular activity. The process of removal of hyalinized tissues and re-vascularization had begun, new collagenous fibres had been formed and undermining resorption locally had already lead to breakthrough of alveolar bone. No differences in extent and localization of vascular and cellular reactions were found between the forces of 50, 100, and 200 cN. Localized areas of root resorption were seen, which seemed to be more prominent with larger forces.

In the case studied after 21 days little activity was observed; no cell-free zones and only few osteoblasts were present, while blood vessels were normal in size but had increased in number. The collagenous fibres along the alveolar bone were less densely packed than on the side of the root surface. The width of the periodontal ligament varied between 150-200 μm . Study of serial sections showed small localized areas of resorption of root cementum.

At the end of phase 2, after 25 days, there was an increased number of osteoclasts with direct resorption along the alveolar bone (Fig. 6-4). Locally, root resorption had proceeded into the dentine. At some spots reparative cementum was deposited. The number of resorbing and depositing cells showed great local differences. Cell-free areas were not present and small vessels penetrated the areas of repair. The attachment of the Sharpey's fibres at the root surface was lost and collagen type III fibres were arranged parallel to the root surface. They were more densely packed in the bone related part of the periodontal ligament than in the tooth related part.

In phase 3, after 81 days, osteoclasts were active directly on the bony bumpers, that protruded into the periodontal ligament space, whereas in the marrow spaces no osteoclasts were present (Fig. 6-5). There was no undermining resorption. Remodelling of the alveolar bone was indicated by the activity of osteoblasts.

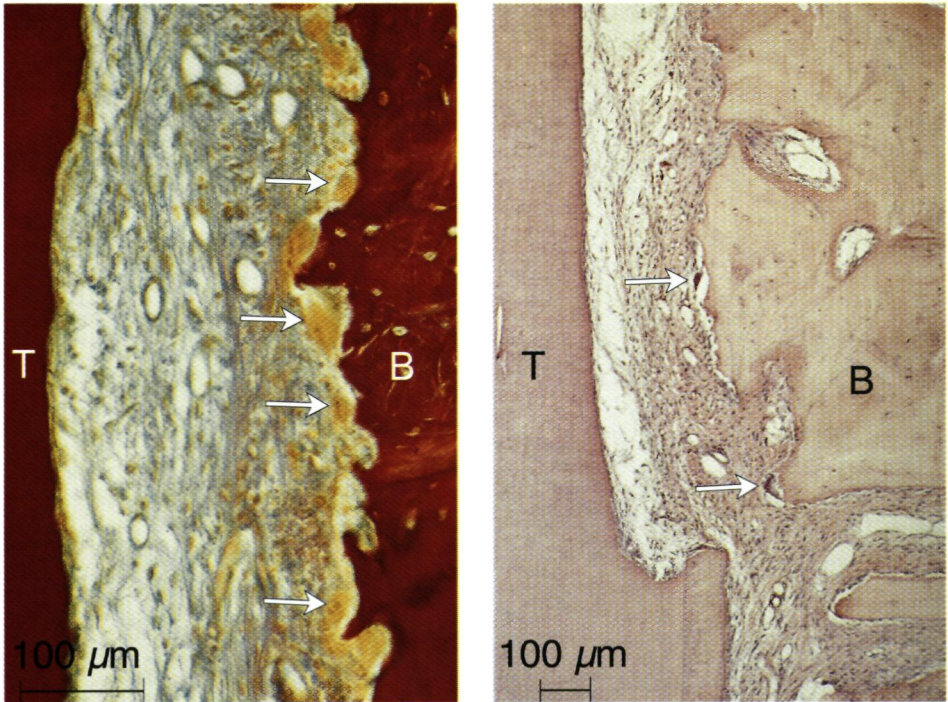


Figure 6-4: *Photomicrograph showing the periodontal ligament at the pressure side of a second premolar to which a force of 200 cN was applied for 25 days. The collagenous fibres grossly are oriented parallel to the root.*

*T = Tooth; B = Alveolar bone; arrows indicate osteoclasts.
Herovici's pentachrome; neg magn x 63.*

Figure 6-5: *Photomicrograph showing the periodontal ligament at the pressure side of a second premolar to which a force of 100 cN was applied for 81 days. Clearly demarcated root resorption is obvious. Note that the width of the periodontal ligament is more or less normal in that area.*

*T = Tooth; B = Alveolar bone; arrows indicate osteoclasts.
Haematoxylin and Eosin; neg magn x 25.*

There were no cell-free areas and the collagen type III fibres were less densely packed at the root side than at the bone side. The fibres were oriented parallel to the root surface and Sharpey's fibres were absent. Extreme resorption of the middle part of the root was found: in some cases about 40% of the width from cementum to pulp had disappeared (Fig. 6-5). The apical one third of the root showed only minimal resorption. No differences in any respect between the 50 cN and 100 cN force samples were found. The width of the periodontal ligament varied between 100-150 μm .

In phase 4 after 119 days with 200 cN, the histologic findings are the same as for the 50 cN and 100 cN samples after 81 days. There was direct resorption and there were no cell-free areas nor undermining resorption. Further extension of root resorption was noticed. Resorption of the alveolar bone had proceeded and the periodontal ligament showed to be continuous with the marrow spaces of the trabecular alveolar bone.

6.4.2 Active tooth movement, tension side

In phase 1 after 7 days, the number of osteoblasts was increased on the bone surface in all cases, resulting in deposition of osteoid around newly formed collagenous fibres (Fig. 6-6). This layer was most readily deposited in the 200 cN sample with a thickness of approximately 50 μm . Active deposition of reparative cementum on the root surface could only be detected in the case of 200 cN. The course of the collagenous fibres was normal, except in the side with 50 cN, where fibres were arranged parallel to the root surface. The width of the periodontal ligament was hardly larger than on the compression side and varied between 150 and 200 μm .

In phase 2, after 14 days, the number of osteoblasts was clearly elevated, and they were regularly arranged along the alveolar bone (Fig. 6-7). A layer of 30-40 μm osteoid was deposited with 50 cN, 50-75 μm with 100 cN, and about 100 μm with 200 cN. Cementoblasts had deposited a thin layer of cementum along the root surface in all cases. Sharpey's fibres, consisting of type I collagen, were embedded in the osteoid and in the cementum (Fig. 6-7). Collagenous fibres appeared to be stretched although the width of the periodontal ligament was hardly larger than normal. Blood vessels seemed to be larger in size than at the pressure side.

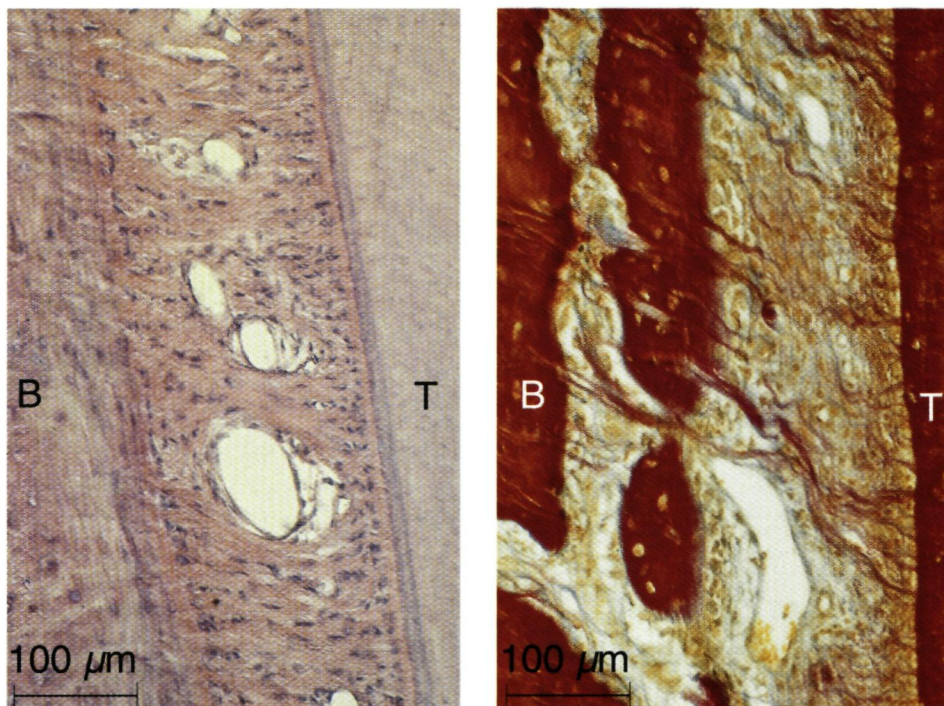


Figure 6-6: *Photomicrograph showing the periodontal ligament at the tension side of a second premolar to which a force of 100 cN was applied for 7 days.*

T = Tooth; B = Alveolar bone;

Haematoxylin and Eosin; neg magn x 63.

Figure 6-7: *Photomicrograph showing the periodontal ligament at the tension side of a second premolar to which a force of 200 cN was applied for 25 days. Sharpey's fibres in the bone and cementum are obvious.*

T = Tooth; B = Alveolar bone;

Herovici's pentachrome; neg magn x 63.

In the case studied after 21 days hardly any osteoid or cementum was deposited, and the fibres were normally arranged. After 25 days, trabecular bone was deposited against the original lamellar bone. Sharpey's fibres extended into the newly formed finger-like bony protrusions, which had a length of about 200 μm and were oriented in the direction of the stretched fibres. A cementum layer of about 30 μm was deposited along the root surface. Large blood vessels were present. The width of the periodontal ligament was about 200 μm and did not differ much from the pressure side.

In phase 3 after 81 days, bone deposition of uniform thickness from the apical to cervical region along the original alveolar bone lamina was demarcated by a reversal line. Numerous osteoblasts were active and showed an epithelioid arrangement (Fig. 6-8). A cementum layer of about 125 μm was deposited along the entire root surface, with increasing thickness in apical direction. The density of the collagenous fibres in the periodontal ligament at the bone surface was less than at the root surface. In some areas collagen type III fibres were arranged parallel to the root surface and Sharpey's fibres were absent. The width of the periodontal ligament was widened to 200-250 μm . Many small blood vessels were seen which were flattened between the collagenous fibres. No differences were found between 50 cN and 100 cN.

In phase 4 after 119 days with 200 cN, cellular cementum was found along the root surface. On the bone side many osteoblasts were depositing trabecular bone (Fig. 6-9), in which Sharpey's fibres were embedded.

6.4.3 Relapse period, former pressure side

Eighteen days after removal of elastics, at the apical and cervical part of the periodontal ligament, still some osteoclasts were present on the former pressure side. No cell-free areas were present and the attachment of the collagenous fibres to the bone was not yet restored.

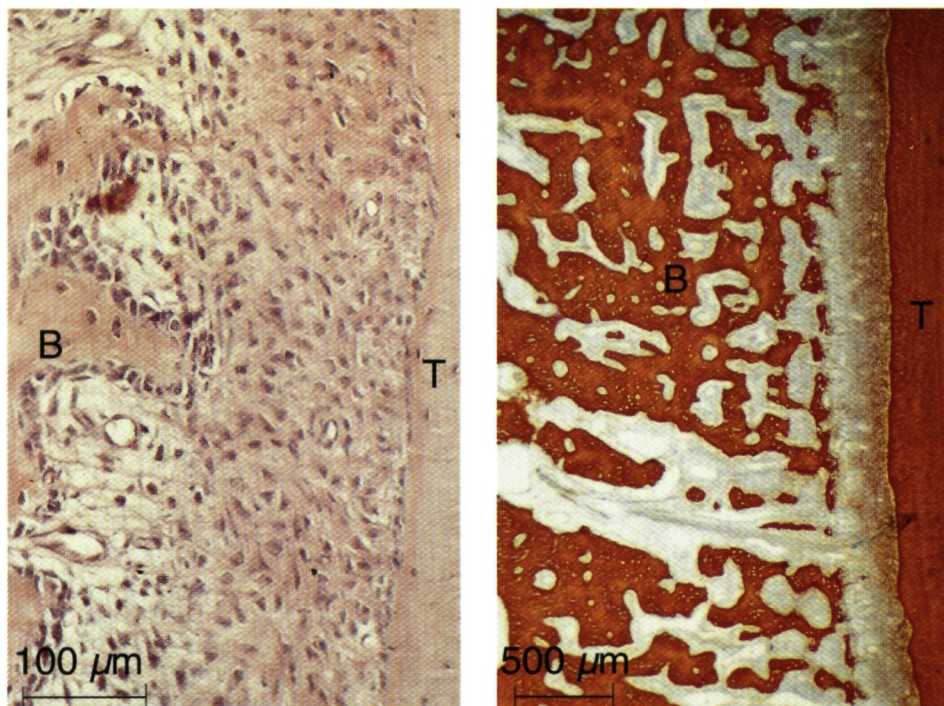


Figure 6-8: *Photomicrograph showing the periodontal ligament at the tension side of a second premolar to which a force of 50 cN was applied for 81 days.*

T = Tooth; B = Alveolar bone;

Haematoxylin and Eosin; neg magn x 63.

Figure 6-9: *Photomicrograph showing the periodontal ligament at the tension side of a second premolar to which a force of 200 cN was applied for 119 days.*

T = Tooth; B = Alveolar bone;

Herovici's pentachrome; neg magn x 10.

In the middle third part, the structure had changed considerably. Osteoblasts had differentiated and first bone deposition was apparent. Reorientation of periodontal ligament fibres had taken place and first Sharpey's fibres were embedded in the alveolar bone (Fig. 6-10). The width of the periodontal ligament was uniform, measuring about 150-200 μm .

At the end of the relapse period two sides which had forces of 50 and 100 cN were studied. In both cases the structure of the periodontal ligament was still somewhat disorganized. Bone deposition had changed the surface of the bone in such a way that it now accurately followed the irregular contour of the root surface which resulted from root resorption during active tooth movement. Cellular activity was normal and in one case a small layer of acellular cementum was deposited on the root surface. In the other case irregular layers of reparative cellular cementum were deposited, resulting in a smoothing of the root outline.

6.4.4 Relapse period, former tension side

Eighteen days after removal of the elastics still some osteoblasts were present on the former tension side, especially in the apical area of the periodontal ligament. Also some root resorption was seen in that area. In the middle part of the periodontal ligament, the structure had changed considerably. The principle fibres had disappeared and mainly thin collagen type III fibres were oriented parallel to the surfaces of bone and root. Osteoclastic bone resorption had resulted in an irregular surface of the alveolar bone (Fig. 6-11). In some cases localized root resorption was also found. The periodontal ligament was about 100 μm in width.

At the end of the relapse in both cases normal cellular activity was found, but the blood vessels seemed smaller than on the former pressure side. Small spots with active resorption were only seen in the apical part of the root. Collagenous fibres in the periodontal ligament were arranged normally.

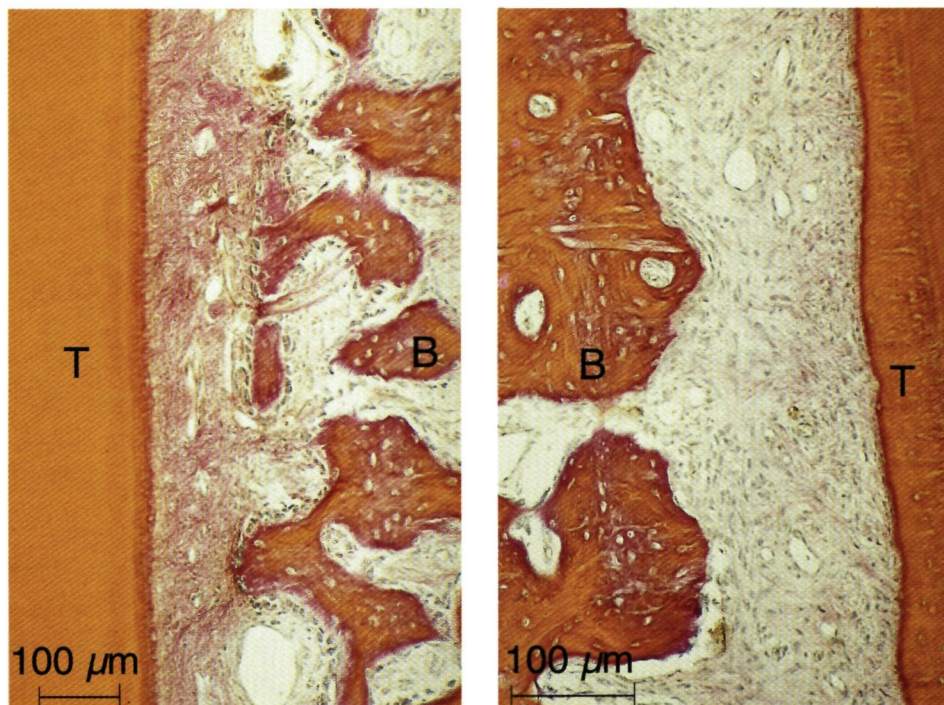


Figure 6-10: *Photomicrograph showing the periodontal ligament at the former pressure side after a relapse period of 18 days. Note the similarity with Fig. 6-5.*

T = Tooth; B = Alveolar bone.

Herovici's pentachrome; neg magn x 40.

Figure 6-11: *Photomicrograph showing the periodontal ligament at the former tension side after a relapse period of 18 days. Note the similarity with Fig. 6-7.*

T = Tooth; B = Alveolar bone.

Herovici's pentachrome; neg magn x 40.

6.5 Discussion

Several factors determine the reaction of a tooth and its surrounding tissues to an orthodontic force. The initial movement of a tooth is to be considered as a bio-elastic reaction and it is mainly dependent on the biophysical characteristics of the periodontal ligament (Bien, 1966; Kardos and Simpson, 1980). However, the timing of tissue reaction to orthodontic forces is species dependent. In adult humans the initial tissue reaction starts within 2 days after force application, while in adult rats reactions are seen within 30 minutes (Rygh, 1973). Besides, the rate of tissue response is age dependent (Reitan, 1954).

Although the structure of the periodontal ligament differs between humans and dogs (Bartley *et al.*, 1970; Reitan and Kvam, 1971), and although the alveolar bone in dogs is denser than in humans, it is likely that in young adult dogs the initial tissue reaction takes place within the first few days of force application (Pilon *et al.*, 1996).

The initial reaction is soon followed by a second reaction in which additional cellular activity is involved. The intermingling of these two reactions starts in juvenile humans 30-36 hours after force application (Reitan, 1951).

In this histological study, the earliest observations were made 7 days after force application, so tissue responses were already under way for some time. At the pressure side, the attachment of Sharpey's fibres to the bone was lost in areas of direct resorption, while only small resorption lacunae were present at the root surface without loss of fibre attachment. The reason for this difference in resorptive activity at the bone and root surface remains unexplained. If a pressure gradient in the interstitial fluid pressure would exist, it is doubtful that it is responsible for the differential osteoclastic activity at the bone and root side, as the same reactions were found in all force groups. As tooth movement continued, root resorption increased, especially in the middle part of the root. This may be explained by the form of the periodontal ligament, which resembles the form of a sandglass. The width in the middle part of the periodontal ligament is smaller than in the cervical and apical part (Boyle, 1955). Therefore compression on the

pressure side in the periodontal ligament is relatively larger in the middle part than in the apical and cervical parts. This differential resorption pattern was present in all phases of active tooth movement. The sandglass form of the periodontal ligament persisted, most likely because tipping movements were impossible, with the used orthodontic appliance. A relationship seemed to exist between the amount and duration of tooth movement on the one hand and the amount of root resorption on the other hand.

At the tension side another picture was found. There, osteoblasts were found along the bone surface. They might partly be re-activated osteoblasts, which were already present in the area, but partly they are probably the result of proliferation and differentiation of mesenchymal cells (Tayer *et al.*, 1968). They deposited osteoid around newly formed collagenous fibres. The gross fibrous structure of the periodontal ligament was undisturbed at the tension sides. Only in the highest force group, a reaction at the root surface was found in the form of the deposition of some new cementum. Also the deposition of osteoid seemed to increase with larger forces.

After this initial phase, a phase of arrest of tooth movement was found, in which prominent changes were found at the pressure side. There, in all force groups, hyalinized areas were present at the middle one third of the periodontal ligament. In these cell-free zones, the periodontal fibrous structures were completely lost, and in the mean time osteoclasts had differentiated in the marrow spaces of the adjacent alveolar bone, starting undermining resorption. Apparently this process was already on the way for some time, as re-vascularization, fibroblast migration, and formation of new collagenous fibres had already started at the borders of the hyalinized areas. The fact that no differences were found in the extent of the hyalinization areas between the different force levels was surprising. Since the extensive research of Reitan (1960, 1967) and Rygh (1972, 1973), it is known that hyalinization of the periodontal ligament starts in the most compressed areas. However, under the given circumstances, compression might be more dependent on biophysical characteristics of the periodontal ligament than on force magnitude.

At the tension side it appeared that the differentiation of osteoblasts continued. The amount of bone deposition, however, was only small, and

dependent of the force magnitude. A thin layer of cementum was deposited at the root surface in all force groups, and the structure of the periodontal collagenous fibres remained normal.

At the end of the phase of tooth arrest, the hyalinized areas had disappeared at the pressure side. The undermining resorption had resulted in localized widening of the periodontal space and in other areas direct osteoclastic bone resorption took place. In the former hyalinized areas, tissue structure was still disorganized and Sharpey's fibres were absent. No deposition of mineralized tissues was found at the surface of the root, nor at the bone surface. So the attachment of the tooth to the alveolar bone was lost and could not be restored. It is not clear if this is related to the orientation of the fibres at the pressure side, which is mainly parallel to the root surface.

At the tension side, on the other hand, the osteoblasts increased their activity and the rate of bone deposition, reflected by a change from lamellar to trabecular bone deposition. During this phase, clearly a transition from a reparative process to an adaptive process took place. The speed of the cellular reactions probably was increased to its biological maximum, which is reflected in increasing rate of tooth movement.

In the following phase, in which the teeth moved with a constant rate, one can assume that the process of remodelling in the periodontal ligament has reached its maximal biological capacity. It is not known which of the activities at the pressure side or the tension side is the limiting factor in determining the rate of tooth movement. The phenomena at both sides remained basically the same. Most attention was drawn by the very local cellular activity at the pressure side: only at the most prominent bony protrusions, where stress concentrations most likely are high, direct resorption was seen. Obviously, in a continuous sequence at different positions along the alveolar wall osteoclasts appeared, and small bony parts were removed as soon as they protruded. This means that the concept of an intermittent tooth movement, where periods of standstill and sudden irregular jumps forward alternate, is not valid any more in this phase of continuous tooth movement, but that the tooth moves smoothly through the surrounding tissues at a constant rate.

At the start of the relapse period a delayed reaction was observed in the

periodontal ligament. According to Reitan and Kvam (1971), in humans it takes 3-4 days before the process of bone resorption is reversed into a process of bone apposition. The results of the present study were not contradictory as after 18 days of relapse, bone resorption was found at the former tension side, and osteoblasts had differentiated or were re-activated at the former pressure side.

The mechanical situation in the periodontal ligament during relapse seemed to be essentially the reverse of that during active tooth movement. This was stressed not only by the fact that the patterns of bone resorption had reversed, but also by the fact that root resorption was encountered at the former tension side.

The process of orthodontic tooth movement is complicated because it is a function of externally applied forces and internally operating biological phenomena. During tooth movement, physiological processes try to cope with the externally applied forces, but pathological reactions at the root surface cannot be avoided. The results indicate that the physiological processes are aiming at the elimination of the noxious influences of the force. On the tension side this attempt appears to be successful, as the structure of the periodontal ligament remains intact, but at the pressure side the reaction is not sufficient and as a consequence other mechanisms have to be activated. Obliteration or compression of blood vessels by this inadequate reaction causes hyalinization in those areas in which the periodontal ligament is narrowest, i.e. at the middle one third of the root. The outcome of this process is a disturbance of the periodontal attachment of a tooth during the entire orthodontic therapy. Furthermore, root resorption seems to be inevitable, not only active tooth movement during treatment, but also during relapse.

6.6 Literature

- BARTLEY MH, TAYLOR GN, JEE WS (1970). Teeth and mandible. In: Andersen AC (ed). The beagle as an experimental dog. Ames, Iowa: The Iowa State University Press, pp. 189-215.
- BIEN SM (1966). Hydrodynamic damping of tooth movement. J Dent Res 45: 907-914.

- BOYLE PE (1955). Influence of function upon teeth and surrounding structures. Arrangement and function of the fibers of the periodontal ligament. In: Kronfeld's histopathology of the teeth and their surrounding structures. Philadelphia (USA): Lea and Febiger, pp. 320-341.
- BURSTONE CJ (1962). Biomechanics of tooth movement. In: Kraus BS, Riedel RA (eds). Vistas in orthodontics. Philadelphia (USA): Lea and Febiger, pp. 197-213.
- BURSTONE CJ (1989). The biophysics of bone remodeling during orthodontics - optimal force considerations. In: Norton LA, Burstone CJ (eds). The biology of tooth movement. Boca Raton, Florida (USA): CRC Press, pp. 321-334.
- DAVIDOVITCH Z, NICOLAY O, ALLEY K, ZWILLING B, LANESE B, SHANFELD JL (1989). First and second messenger interactions in stressed connective tissue in vivo. In: Norton LA, Burstone CJ (eds). The biology of tooth movement. Boca Raton, Florida (USA): CRC Press, pp. 97-130.
- KARDOS T, SIMPSON L (1980). A new periodontal membrane biology based upon thixotropic concepts. *Am J Orthod* 77: 508-515.
- KING GJ, FISCHLSCHWEIGER W (1982). The effect of force magnitude on extractable bone resorptive activity and cemental cratering in orthodontic tooth movement. *J Dent Res* 61: 775-779.
- KUITERT RB (1988). Histologische veranderingen in het parodontale ligament en de gingiva gedurende de eerste 24 uur van orthodontische tandverplaatsing bij konijnen. PhD Thesis, University of Amsterdam, The Netherlands, pp. 185-249.
- LEVAME M, MEYER F (1987). Le picropolychrome de Herovici: application a l'identification des collagènes de types I et III. *Path Biol* 35: 1183-1188.
- PILON JJGM, KUIJPERS-JAGTMAN AM, MALTHA JC (1996). Magnitude of orthodontic forces and rate of bodily tooth movement - An experimental study in beagle dogs. *Am J Orthod Dentofac Orthop* 110: 16-23.
- REITAN K (1951). The initial tissue reaction incident to orthodontic tooth movement as related to the influence of function. An experimental histologic study on animal and human material. *Acta Odont Scand* 9: Suppl. 6.
- REITAN K (1954). Tissue reaction as related to the age factor. *Dent Record* 74: 271-278.
- REITAN K (1960). Tissue behaviour during orthodontic treatment. *Am J Orthod* 46: 881-899.
- REITAN K (1964). Effects of force magnitude and direction of tooth movement on different alveolar bone types. *Angle Orthod* 34: 244-255.

- REITAN K (1967). Clinical and histologic observations on tooth movement during and after orthodontic treatment. *Am J Orthod* 53: 721-745.
- REITAN K (1969). Principles of retention and avoidance of posttreatment relapse. *Am J Orthod* 55: 776-790.
- REITAN K, KVAM E (1971). Comparative behaviour of human and animal tissue during experimental tooth movement. *Angle Orthod* 41: 1-14.
- RYGH P (1972). Ultrastructural cellular reactions in pressure zones of rat molar periodontium incident to orthodontic tooth movement. *Acta Odont Scand* 30: 575-593.
- RYGH P (1973). Ultrastructural changes in pressure zones of human periodontium incident to orthodontic tooth movement. *Acta Odont Scand* 31: 109-122.
- RYGH P, BOWLING K, HOVLANDSDAL L, WILLIAMS S (1986). Activation of the vascular system: a main mediator of periodontal fiber remodelling in orthodontic tooth movement. *Am J Orthod* 89: 453-468.
- SANDSTEDT C (1904). Einige Beitrage zur Theorie der Zahnregulierung. *Nord Tandlaek Tidsskr* 5: 236-256.
- SCHWARZ AM (1932). Tissue changes incident to tooth movement. *Int J Orthod* 18: 331-352.
- STOREY E (1973). The nature of tooth movement. *Am J Orthod* 63: 292-314.
- STUTEVILLE OH (1938). Injuries caused by orthodontic forces and the ultimate result of these injuries. *Am J Orthod Oral Surg* 24: 103-116.
- TAYER BH, GIANELLY AA, RUBEN MP (1968). Visualization of cellular dynamics associated with orthodontic tooth movement. *Am J Orthod* 54: 515-520.
- YAMASAKI K (1989). Pharmacological control of tooth movement. In: Norton LA, Burstone CJ (eds). *The biology of tooth movement*. Boca Raton, Florida (USA): CRC Press, pp. 287-320.

Chapter 7

General discussion

7.1 Introduction

Reseachers as well as clinicians in the field of orthodontics are interested in the development of treatment strategies producing optimal biologic responses, which means maximum rate of tooth movement with minimal irreversible damage to the root, periodontal ligament, and alveolar bone.

Orthodontic forces cause complex changes in the stress and strain distribution within the periodontal ligament. These changes are dependent on root form, height of the alveolar crest, geometry and biomechanical characteristics of alveolar bone and periodontal ligament, and on the type of force and subsequent tooth movement (Burstone, 1962). The relationship between these parameters and changes in stress and strain distribution during tooth movement however is largely unknown.

The purpose of our study was to get more insight into the relationship between orthodontic forces and the rate of subsequent bodily tooth movement. At the start of this study it was not clear whether a dose-response relation exists between the magnitude of orthodontic forces and the long term tissue responses in the periodontal ligament and alveolar bone. The characteristics of relapse after active tooth movement were also subject of this study.

7.2 Standardization of the experimental setting

7.2.1 *Animal model*

Beagle dogs were used in this experiment because the anatomy and structure of the periodontal ligament and alveolar bone show a large resemblance with that of humans (Bartley *et al.*, 1970; Hull *et al.*, 1974). As humans, beagles are susceptible to periodontal diseases, but they show some different reactions compared to humans. Beagles show more vascular proliferation, less fibrosis, and most important no pocket formation. When the alveolar bone level moves apically due to periodontal disease, the gingiva retracts simultaneously in the same direction (Page and Schroeder, 1981). Strict hygienic measures were taken in our experiment to keep the gingiva as healthy as possible and to

prevent loss of alveolar bone. Histologic study of the periodontal tissue showed good gingival condition and a normal bone level in the furcation area throughout the experiment in all dogs.

Spontaneous migration of teeth, adjacent to the extraction site, had to be determined because this could interfere with tooth movement during the actual experiment. Teeth tend to move into the direction of an extraction site in humans and monkeys (Richardson, 1965; Cookson, 1971). Measurements on series of dental casts of beagle dogs, however, showed that all teeth moved away from the extraction site. This may be explained by growth of the mandible with drifting of teeth, or by eruptive movements of mandibular teeth which have diverging inclinations.

In our experiments the second lower premolars were moved distally into an edentulous alveolar area. In a study, in beagle dogs, on tooth movement into an area in which alveolar bone height was surgically reduced, Lindskog-Stokland *et al.* (1993) found no loss of connective tissue attachment, while the histological pocket depth remained unaltered by the treatment. It has also been shown by Reed *et al.* (1985) that orthodontic tooth movement into an extraction site has no detrimental effect upon the long-term periodontal status.

7.2.2 Force system

During orthodontic tooth movement, physiological forces, such as those from muscular activity, are always acting. These forces contribute to the total force system which is determined by magnitude, direction, moment to force ratio, duration, frequency, load-deflection curve, and range of activation. An accurate description of the total force system, however, is impossible because especially the physiological forces change continuously.

The orthodontic appliance used in the present study was designed to add a constant and continuous force to the physiological system. This force had to be constant in magnitude and direction and independent of the amount of tooth movement. The teeth in our experiment were forced to move bodily because this type of tooth movement is reproducible in contrast to tipping movements, where varying degrees of crown and root movement complicate interpretation of the results. Another advantage of bodily tooth movement is that stresses in the periodontal ligament are more evenly distributed along the

root surface (Reitan, 1957; Nikolai, 1975). In studying stress profiles within the periodontal ligament Andersen *et al.* (1991) showed, with the aid of an improved finite-element model, that bodily tooth movement produced an almost uniform stress distribution. The periodontal ligament showed a high degree of linearity in behaviour, in contrast to the general belief of a non-linear and anisotropic behaviour. The uniform stress distribution may be cause of the fact that bodily tooth movement reduces the risk of root resorption compared to tipping tooth movement (Reitan, 1974).

Bodily tooth movement can be achieved by a force acting through the centre of resistance. This centre of resistance, however, is difficult to determine (Burstone, 1962) and it depends on root length and alveolar bone height (Tanne *et al.*, 1991). For a multiple rooted tooth it is situated in the region of the furcation (Burstone, 1962).

When elastics are attached to the orthodontic appliance in this study, a couple is inevitably introduced because the hooks are located buccally to the centre of resistance of the second lower premolar. The sliding bar in the orthodontic device, however, prevents tipping movement of the teeth. It is not clear, how much energy, delivered by the elastics, is lost due to frictional resistance in this experiment. In physics, frictional resistance $F_{fr} = F \times U$, where F is the force pressing the surfaces together, and U is the coefficient of friction. The value of U is mainly dependent on the materials which are involved and only slightly affected by other factors, such as the extent of the surface area or speed of movement (Gamow, 1976). Before a tooth can move, static friction within the appliance has to be overcome. During tooth movement, kinetic friction occurs. Andreasen and Quevedo (1970) found that up to 50% of applied force could be dissipated due to friction in an edgewise appliance, while Tidy (1989) even found more than 60%, which may give an indication of the high coefficient of friction for two stainless steel surfaces in general. More specific statements about frictional resistance in our device will demand separate laboratory experiments.

The sliding bar is oriented in a direction parallel to the alveolar process. In this way movement of the roots into the lingual or labial cortical bone plates, which could lead to arrest of tooth movement, is prevented. As was pointed out by Burstone (1982) and Isaacson *et al.* (1993), moment to force

ratios change constantly during tooth movement. This is inherent to the dynamic load deflection rate of any orthodontic device and the physiological conditions. This means that even so-called bodily tooth movement is inevitably the result of constantly changing small tipping movements.

7.2.3 Forces

A major problem in studying the results of previous experiments on tooth movement is that they are difficult to interpret and compare because the definition of the applied orthodontic forces, and the measurement and determination of the type of orthodontic tooth movement, is not uniform or incomplete.

In the present study continuously acting forces of constant magnitude were chosen to produce tooth movement. In clinical orthodontics, however, most forces are interrupted, which means that in between two activations force magnitude declines, possibly even to zero.

Forces in clinical orthodontics are often generated by closing chains. These closing chains are not suitable for our experimental setting because of their high degree of stiffness and their large force decay over time (Kuster *et al.*, 1986). Elastics, which are normally used for intermaxillary forces, were used in this experiment. These elastics are also subject to force degradation but in the first part of the present study it became clear that, if these elastics were stretched in artificial saliva for two days prior to clinical use, they could produce constant forces for about three weeks. Therefore prestretched elastics were used during the experiment and their force levels were measured twice a week. When a deviation of more than 5% of the desired force was found, elastics were replaced.

In literature a large range of forces or pressures is advocated to be optimal for tooth movement (Jarabak and Fizell, 1963; Hixon *et al.*, 1969; Boester and Johnston, 1974; Quinn and Yoshikawa, 1985; Miura *et al.*, 1986). Based on these data, pressure levels of about 10, 20 and 40 kPa were chosen, which can be qualified as low, medium, and high. These pressure levels could be approximated for lower second premolars in beagles using forces of 50, 100, and 200 cN.

7.3 The optimal force theory

Several hypotheses have been framed for the relationship between rate of tooth movement and stress in the periodontal ligament. Andreasen and Johnson (1967), and Hixon *et al.* (1970) showed that changing stress causes a change in rate of tooth movement. Larger forces produced more tooth movement. Boester and Johnston (1974), and Hixon *et al.* (1969), however, found no measurable increase in rate of tooth movement above certain force levels, which means that a maximum rate of tooth movement may exist. Storey and Smith (1952) originally proposed a reversal in rate of tooth movement above a certain force level, but analysis of their data reveals no evidence for such a phenomenon (Quinn and Yoshikawa, 1985). Also the conclusion that an optimal force level exists to produce a maximum rate of bone remodelling is not justified by the data that are presented by Storey in 1955. The findings of Burstone and Groves (1961) and Andreasen and Zwanziger (1980) support the existence of a maximum rate of tooth movement. Owman-Moll *et al.* (1996a) found no difference in amount of tipping tooth movement in human premolars between forces of 50 cN and 100 cN after 7 weeks. However, the mean amount of tooth movement increased 50% when a force of 200 cN was applied. According to Quinn and Yoshikawa (1985), a linear relationship up to a maximum seems to be most probable, where further increase of stress causes no change in the rate of tooth movement.

Our findings seem to exclude a linear relationship because no significant differences were found in the mean rate of tooth movement between the three experimental force groups. Also for the anchorage unit, in which stress was estimated to be about 10 times smaller than for the second premolar, no significant differences were found in the mean rate of tooth movement between the force groups.

Stress in the periodontal ligament was estimated to be 1, 2, and 4 kPa in the anchorage unit and 10, 20, and 40 kPa for the second premolar. For these two groups of pressure we have found two plateaus with different rates of tooth movement. The rate of tooth movement below stresses of 1 kPa has yet to be determined. Weinstein *et al.* (1963) found that forces as small as

4 g, which produce a stress of approximately 0.2 kPa, can move teeth at a rate of 0.1 mm per week.

Generally, it can be concluded that no optimal force magnitude, or better, no optimal pressure magnitude can be advised. Within each force group, slow movers and fast movers were present. Maximal rate of tooth movement was the same in all force groups, which indicates that even with the lightest pressure in our experiment maximal biological capacity was already reached. In other animal studies large individual differences in amount or rate of tooth movement have been reported (Reitan, 1967; Buck and Church, 1972; Mitchell *et al.*, 1973). A large variation in response between individuals was also found in humans (Hixon *et al.*, 1970; Burstone and Groves, 1961; Owman-Moll, 1996a,b). The most probable explanation for this variation is that individual differences exist in the metabolic capacity, which determines the rate of bone turn-over and the connective tissue reaction. Bone density and the number of cells which are involved in the remodelling of the periodontal ligament are supposed to be important factors in the process of tooth movement (Reitan, 1957), but also other factors as cytokines and growth factors may be responsible for individual differences (Davidovitch *et al.*, 1988).

7.4 Relapse

Relapse after orthodontic treatment is an important clinical problem, which has been related to local factors in the periodontal ligament and alveolar bone, and general factors as growth and function (Reitan, 1969). For a proper understanding of the mechanisms that are involved in the process of relapse, time-displacement curves were made of the relapse period. Relapse was studied directly after a period of active movement of 122 days, without a period of retention. The outcome of this part of the study offers data for future research on effectiveness of different retention regimes.

The mean amount of relapse was almost 40% of the active tooth movement and mean duration was 78 days. These findings stress the necessity for a certain retention period after orthodontic treatment. It is

surprising that the curves, representing the long term recovery of the tooth surrounding structures during relapse, suggest viscoelastic properties. They show the same characteristics as the immediate short time recovery after peak force application (Kardos and Simpson, 1979, 1980). Immediate relapse is attributed to movement of the tooth in its socket after removal of the orthodontic force. The process of long term recovery, however, is far more complicated than the immediate recovery, since it involves complex cellular activity.

Large individual differences were found for the amount and duration of relapse, which is in concordance with our findings during active tooth movement. The left and right side of each dog were highly correlated with respect to the amount of relapse and the half-way point of the curve, irrespective of the force magnitude. This indicates that not only active tooth movement but also the process of relapse is individually "predetermined", as could be expected since the same biologic mechanisms are involved. The significant correlation found between the amount of active tooth movement, and the amount of relapse and duration of the relapse period, indicates that the amount of active tooth movement should be taken into account when a retention regime is chosen. Retention measures should concentrate on removal of the information, that is stored in some way in the biologic system of the periodontal ligament, producing this unwanted tooth movement.

7.5 Histologic findings

During the initial movement of a tooth in its socket, in all experimental animals direct resorptive activity was found with a differential activity at the bone and root side. Although there is no explanation for this phenomenon, a pressure gradient may be involved. With a three-dimensional finite element stress analysis, Tanne *et al.* (1987) found stress to be highest in the root related area, intermediate in the alveolar bone related area, and smallest in the periodontal ligament proper. Because in all force groups, with different pressure values, the same reactions were observed, a pressure gradient alone is probably not responsible for this differential activity of osteoclasts.

The basic form of the time-displacement curves appeared to be the same for all experimental sides. In most cases, initial movement was followed by a period of standstill. Mean duration of this phase was 7 days in all force groups. The large individual differences in duration, ranging from 0 to 35 days, as found in the present study are in concordance with the findings of Reitan (1967). He found hyalinization periods varying between 5 days and 2 months, the latter in dogs with "high bone density". It was surprising that no differences in the mean extent of the hyalinization areas between the three force groups were found. This may be related to the fact that no differences in mean duration of this phase were found between the force groups.

Compression of the periodontal ligament was found at all pressure sides and some widening at the tension sides. It seems that the resorption of bone at the pressure side is the delaying factor in the process of tooth movement. During the following phase of tooth movement, bone deposition at the tension side changes from lamellar to trabecular, reflecting increased cellular activity. The fact that in all force groups the same maximum rate of tooth movement was found, indicates that biologic processes reached their maximum capacity.

Another finding was the very local cellular activity on the pressure side. Only at the most prominent bony protrusions osteoclasts were active. It seemed that osteoclasts were constantly changing their positions along the alveolar wall to be active at newly emerging bony protrusions. During the phase of increasing and linear rate of tooth movement, no cell-free hyalinized areas were present anymore. The concept of intermittent tooth movement, where periods of standstill during hyalinization and tooth movement after a "breakthrough" alternate, appears to be invalid. Unpublished data of daily measurement of tooth movement in this experiment indeed indicate small daily changes in tooth position. Future research with continuous registration of tooth movement is needed to study exactly the process of continuous long term tooth movement.

7.6 Remodelling of the periodontal ligament

The rate of remodelling in the periodontal ligament seems to be the limiting

factor in the process of tooth movement. The importance of vascular proliferation and the presence of macrophages in areas of tension and bone resorption was stressed by Rygh *et al.* (1986). Different techniques have been used experimentally to increase the resorption of bone at the pressure side. Locally applied electric currents (Norton, 1989) and biochemical intervention by injection directly into the periodontal ligament or by systemic administration of different agents have been tested (for a review, see: Yamasaki, 1989). Application of electric currents in animal experiments suggests that bone related cells may be activated slightly and remodelling may be accelerated (Davidovitch *et al.*, 1980; Stark and Sinclair, 1987). Hashimoto (1990) has shown that a micro-pulsed electrical current of 10 μ A during 14 days can stimulate remodelling of alveolar bone and induces more rapid tipping tooth movement in cats. However, the clinical significance of application of electric currents can be questioned, because of irritation of the mucosa at the point of application and the uncertainty about the long term effects on pulpal tissue, periodontal ligament and alveolar bone. The effect of stress-generated piezoelectric currents has been studied by Shapiro *et al.* (1979), but Norton (1989) concluded that these currents may play a role in bone homeostasis but probably not in remodelling during orthodontic tooth movement.

The biological responses to orthodontic forces on the cellular level are not quite understood. It may be that osteoblasts at the pressure side are the principal cells involved (Miyajima *et al.*, 1992). Excitation of stress-sensitive membrane receptors on osteoblasts may result in the activation of Phospholipase A2, an enzyme which initiates the arachidonic acid cascade. One of the families of products of this cascade consists of the Prostaglandins, of which Prostaglandin E2 (PGE2) seems to be the most important one (Sandy *et al.*, 1992). The action of PGE2 seems to be twofold. On the one hand it might stimulate collagenase production by the osteoblasts themselves by an autocrine action (Hamilton *et al.*, 1984, Heath *et al.*, 1984), and on the other hand it might induce the activation or differentiation of osteoclasts by a paracrine action (Sakomoto and Sakomoto, 1985).

Injections of prostaglandins into the periodontal ligament (Yamasaki *et al.*, 1984) accelerate tooth movement. Ashcraft *et al.* (1992) showed that orthodontic tooth movement and relapse significantly increased in animals

which were subjected to corticosteroid-induced osteoporosis. Mediators as bradykinin and thrombin, which are present due to inflammatory reactions after application of orthodontic force, are known to stimulate the prostanoid biosynthesis and thus are involved in tissue reactions during orthodontic tooth movement (Marklund *et al.*, 1994). Administration of indomethacin, an inhibitor of prostaglandin biosynthesis, significantly reduces orthodontic tooth movement in cats (Chumbley and Tuncay, 1986). Inhibition of prostaglandin synthesis by acetyl-salicylic acid has shown that prostaglandins may not be the only mediators of bone resorption associated with tooth movement (Wong *et al.*, 1992).

Lilja *et al.* (1983) found that in rats changes in distribution and activity of enzymes, such as acid phosphatase and lactate dehydrogenase, which are associated with tissue degradation incident to orthodontic tooth movement, are independent of force magnitude.

In conclusion it can be stated that orthodontic tooth movement can probably not be accelerated by changing mechanical treatment regimes but only by controlling local and systemic factors, which are involved in the recruitment and activation of bone resorbing cells.

7.7 Root resorption

Several studies have shown that root resorption can not be avoided during orthodontic tooth movement (Reitan, 1974; Rygh, 1977). Factors affecting root resorption have been divided into biologic factors, mechanical factors, and combined biologic and mechanical factors. An excellent literature review has been made by Brezniak and Wasserstein (1993a,b). The amount of root resorption is also associated with anatomical variations of root form (Newman, 1975; Levander and Malmgren, 1988). Controversial reports have been published on root resorption and alveolar bone density (Wainwright, 1973; Reitan, 1974; Goldie and King, 1984). Also treatment variables such as duration of therapy and use of class II elastics (Linge and Ohm Linge, 1991) seem to affect root resorption. Results from several authors, with respect to the relation between the magnitude of the orthodontic force and the

amount of root resorption, are not uniform. In general, however, there is a tendency to recognize the risk of large forces with respect to root resorption (Rygh, 1977; Harry and Sims, 1982). According to Reitan (1974), also the direction of tooth movement is important: with intrusion and tipping movements root resorptions are seen more often and are more severe than with extrusion or bodily tooth movement. On the other hand, Owman-Moll *et al.* (1994, 1996b) found no differences in root resorption of human premolars, which were tipped buccally with forces of 50 cN, 100 cN, and 200 cN. Furthermore, they found no association between root resorption and amount of tooth movement during a period of 7 weeks.

Many studies have been focusing on apical root resorption, using a radiographic evaluation of root length. Lateral root resorptions often are not included. Because of the anatomy of the root, however, lateral resorptions have to be rather extensive before they become evident on radiographs. In our histological study apical root resorptions were minimal, while lateral root resorptions were far more extensive. Due to the sandglass form of the periodontal ligament (Boyle, 1955), stress in the periodontal ligament at the middle third part of the root may be higher than at the coronal and apical part of the root. This sandglass form did not change during bodily tooth movement in our experiment. An important finding, with respect to lateral root resorptions, is that during translation stress in the periodontal ligament varies somewhat in occluso-apical direction, being highest at the center of the root (Tanne *et al.*, 1987). Normal root anatomy may explain the lower stress found at the apex.

In our study we found small spots of local root resorptions within 7 days after the application of force in all force groups. At the end of the hyalinization period, root resorptions were found to be more extensive with larger forces. After 25 days root resorption had even proceeded into the dentine. Extreme resorption of dentine to 40% of its width was seen after 81 days in the middle part of the root, so there seems to be a relationship between the amount of tooth movement or the duration of therapy and the amount of root resorption. Many investigators believe that root resorption is directly related to the amount of movement (Dermaut and De Munck, 1986; Sharpe *et al.*, 1987). Brudvik and Rygh (1995) have shown that the extent of

root resorption corresponds with the extent of the hyalinized zone. They also found that repair starts from the periphery of the resorbed lacunae, and that root resorption can continue even after active treatment.

It is unknown if continuous forces are the best choice to minimize the amount of root resorption on the long run. Owman-Moll *et al.* (1994) have shown that tipping human premolars buccally was more effective with a continuous force than with an interrupted force during 7 weeks, while no differences in amount and severity of root resorptions were found. Individual variations in number and severity of root resorptions were large for both force regimes. On the other hand, in a clinical experimental study by Levander *et al.* (1994), it was shown that the amount of root resorption was significantly less in patients treated with an interruption of 2 to 3 months than in those treated without interruption.

7.8 Clinical consequences

The present study has shown that large individual differences exist in rate of bodily tooth movement. The rate of tooth movement is not dependent of the applied force. This means that 'slow movers' and 'fast movers' can be distinguished. When this holds true for humans too, the orthodontist should be aware of the fact that increasing force in a certain individual will probably not increase the rate of tooth movement during orthodontic treatment, but may adversely result in more extensive root resorption.

Nowadays, a tendency exists in clinical orthodontics to use low force-deflection wires (Miura *et al.*, 1986). These wires produce forces, which act continuously with a constant force level almost irrespective of wire deflection. Sudden changes in stress levels in the periodontal ligament during tooth movement can be avoided with these wires and initial activation is performed easily without excessive forces. The question is whether this kind of force regime provides optimal biologic responses. These wires may offer a solution for a problem that does not exist, or even worse, may cause unwanted side-effects.

Up to now there is no prove that forces with a constant magnitude

and/or with a continuous action produce more favourable results in terms of rate of tooth movement and damage to the periodontal ligament than intermittent or interrupted forces. It is of great clinical importance that this question is answered in the near future. It seems that the discussion on optimizing biomechanical therapy should not concentrate on changing force magnitude and stress levels in the periodontal ligament, but on increasing metabolic activity and cellular processes that might be responsible for the large inter-individual differences.

7.9 Literature

- ANDERSEN KL, PEDERSEN EH, MELSEN B (1991). Material parameters and stress profiles within the periodontal ligament. *Am J Orthod Dentofac Orthop* 99: 427-440.
- ANDREASEN G, JOHNSON P (1967). Experimental findings on tooth movements under two conditions of applied force. *Angle Orthod* 37: 9-12.
- ANDREASEN GF, QUEVEDO FR (1970). Evaluation of frictional forces in the .022" x .028" edgewise bracket in vitro. *J Biomed* 3: 151-160.
- ANDREASEN GF, ZWANZIGER D (1980). A clinical evaluation of the differential force concept as applied to the edgewise bracket. *Am J Orthod* 78: 25-40.
- ASHCRAFT MB, SOUTHARD KA, TOLLEY EA (1992). The effect of corticosteroid-induced osteoporosis on orthodontic tooth movement. *Am J Orthod Dentofac Orthop* 102: 310-319.
- BARTLEY MH, TAYLOR GN, JEE WS (1970). Teeth and mandible. In: Andersen AC (ed.) *The beagle as an experimental dog*. Ames, Iowa: The Iowa State University Press, pp. 189-215.
- BOESTER C, JOHNSTON L (1974). A clinical investigation of the concepts of differential and optimal force in canine retraction. *Angle Orthod* 44: 113-119.
- BOYLE PE (1955). Influence of function upon teeth and surrounding structures. Arrangement and function of the fibers of the periodontal ligament. In: Kronfeld's *histopathology of the teeth and their surrounding structures*. Philadelphia (USA): Lea and Febiger, pp. 320-341.
- BREZNIAK N, WASSERSTEIN A (1993a). Root resorption after orthodontic treatment: Part 1. Literature review. *Am J Orthod Dentofac Orthop* 103: 62-66.

- BREZNIAK N, WASSERSTEIN A (1993b). Root resorption after orthodontic treatment: Part 2. Literature review. *Am J Orthod Dentofac Orthop* 103: 138-146.
- BRUDVIK P, RYGH P (1995). Transition of orthodontic root resorption-repair sequence. *Eur J Orthod* 17: 177-188.
- BUCK D, CHURCH D (1972). A histologic study of human tooth movement. *Am J Orthod* 62: 507-516.
- BURSTONE CJ, GROVES MH (1961). Tresshold and optimum force values for maxillary tooth movement. *J Dent Res* 39: 695.
- BURSTONE CJ (1962). The biomechanics of tooth movement. In: Kraus BS, Riedel RA (eds.) *Vistas in orthodontics*. Philadelphia (USA): Lea and Febiger, pp. 197-213.
- BURSTONE CJ (1982). The segmented arch approach to space closure. *Am J Orthod* 82: 361-378.
- CHUMBLEY AB, TUNCAY OC (1986). The effect of indomethacin (an asperin-like drug) on the rate of orthodontic tooth movement. *Am J Orthod* 89: 312-314.
- COOKSON A (1971). Space closure following loss of lower first premolars. *Dent Practit* 21: 411-416.
- DAVIDOVITCH Z, FINKELSON MD, STEIGMAN S, SHANFELD JL, MONTGOMERY PC, KOROSTOFF E (1980). Electric currents, bone remodeling, and orthodontic tooth movement. *Am J Orthod* 77: 33-47.
- DAVIDOVITCH Z, NICOLAY OF, NGAN PW, SHANFELD JL (1988). Neurotransmitters, cytokines, and the control of alveolar bone remodeling in orthodontics. *Dent Clin North Am* 32: 411-435.
- DERMAUT LR, DE MUNCK A (1986). Apical root resorption of upper incisors caused by intrusive tooth movement: a radiographic study. *Am J Orthod Dentofac Orthop* 90: 321-326.
- GAMOW C (1976). *Physics: foundations and frontiers*. 3rd ed. New Jersey: Prentice-Hall, p. 25.
- GOLDIE RS, KING GJ (1984). Root resorption and tooth movement in orthodontically treated, calcium-deficient, and lactating rats. *Am J Orthod* 85: 424-430.
- HAMILTON JA, LIGELBACH SR, PARTRIDGE NC, MARTIN TJ (1984). Stimulation of plasminogen activator in osteoblast-like cells by bone resorbing hormones. *Biochem Biophys Res Comm* 122: 230-236.

- HARRY MR, SIMS MR (1982) Root resorption in bicuspid intrusion a scanning electromicroscopic study *Angle Orthod* 52 235-258
- HASHIMOTO H (1990) Effect of micropulsed electricity on experimental tooth movement *Nippon Kyosei Shika Gakkai Zasshi* 49 352-361
- HEATH JK, ATKINSON SJ, MEIKLE MC, REYNOLDS JJ (1984) Mouse osteoblasts synthesize collagenase in response to bone resorbing agents *Biochem Biophys Acta* 802 151-154
- HIXON E, ATIKIAN H, CALLOW GE, McDONALD HW, TACY RJ (1969) Optimal force, differential force, and anchorage *Am J Orthod* 55 437-457
- HIXON E, AASEN TO, ARANGO J, CLARK RA, KLOSTERMAN R, MILLER SS, ODAM WM (1970) On force and tooth movement *Am J Orthod* 57 476-489
- HULL PS, SOAMES JV, DAVIES RM (1974) Periodontal disease in a beagle dog colony *J Comp Pathol* 84 143-150
- ISAACSON RJ, LINDAUER SJ, DAVIDOVITCH M (1993) On tooth movement *Angle Orthod* 63 305-309
- JARABAK JR, FIZZELL JA (1963) Technique and treatment with light-wire appliances, light differential forces in clinical orthodontics St Louis (USA) CV Mosby Comp , pp 259
- KARDOS TB, SIMPSON LO (1979) A theoretical consideration of the periodontal membrane as a collagenous thixotropic system and its relationship to tooth eruption *J Period Res* 14 444-451
- KARDOS TB, SIMPSON LO (1980) A new periodontal membrane biology based upon thixotropic concepts *Am J Orthod* 77 508-515
- KUSTER R, INGERVALL B, BURGIN W (1986) Laboratory and intra-oral tests of the degradation of elastic chains *Eur J Orthod* 8 202-208
- LEVANDER E, MALMGREN O (1988) Evaluation of the risk of root resorption during orthodontic treatment a study of upper incisors *Eur J Orthod* 10 30-38
- LEVANDER E, MALMGREN O, ELIASSON S (1994) Evaluation of root resorption in relation to two orthodontic treatment regimes A clinical experimental study *Eur J Orthod* 16 223-228
- LILJA E, LINDSKOG S, HAMMERSTROM L (1983) Histochemistry of enzymes associated with tissue degradation incident to orthodontic tooth movement *Am J Orthod* 83 62-75

- LINDSKOG-STOKLAND B, WENNSTRÖM JL, NYMAN S, THILANDER B (1993). Orthodontic tooth movement into edentulous areas with reduced bone height. An experimental study in the dog. *Eur J Orthod* 15: 89-96.
- LINGE L, OHM LINGE BO (1991). Patient characteristics and treatment variables associated with apical root resorption during orthodontic treatment. *Am J Orthod Dentofac Orthop* 99: 35-43.
- MARKLUND M, LERNER UH, PERSSON M, RANSJÖ M (1994). Bradykinin and thrombin stimulate release of arachidonic acid and formation of prostanoids in human periodontal ligament cells. *Eur J Orthod* 16: 213-221.
- MITCHELL DL, BOONE RM, FERGUSON JH (1973). Correlation of tooth movement with variable forces in the cat. *Angle Orthod* 43: 154-161.
- MIURA F, MASAKUNI M, OHURA Y, KARIBE M (1986). The super-elastic property of the Japanese NiTi alloy wire for use in orthodontics. *Am J Orthod Dentofac Orthop* 90: 1-10.
- MIYAJIMA K, KASAI R, KAHN AJ, HAYAKAWA T, IIZUKA T (1992). Biologic mechanisms of tooth movement; in vitro analysis and clinical application. In: Davidovitch Z (ed.) *The biological mechanisms of tooth movement and craniofacial adaptation*. Columbus, Ohio (USA): The Ohio State Univ., College of Dentistry, pp. 311-317.
- NEWMAN WG (1975). Possible etiologic factors in external root resorption. *Am J Orthod* 67: 522-39.
- NIKOLAI RJ (1975). An optimum orthodontic force theory as applied to canine retraction. *Am J Orthod* 68: 290-302.
- NORTON LA (1989). Stress generated potentials and bioelectric effects: their possible relationship to tooth movement. In: Norton LA, Burstone CJ (ed.) *The biology of tooth movement*. Boca Raton, Florida (USA): CRC Press Inc., pp. 349-357.
- OWMAN-MOLL P, KUROL J, LUNDGEN D (1994). Continuous versus interrupted continuous orthodontic force related to early tooth movement and root resorptions. An intra-individual study in adolescents. *Angle Orthod* (accepted for publication 1994).
- OWMAN-MOLL P, KUROL J, LUNDGEN D (1996a). Effects of a doubled orthodontic force magnitude on tooth movement and root resorptions. An inter-individual study in adolescents. *Eur J Orthod* 18: 141-150.
- OWMAN-MOLL P, KUROL J, LUNDGEN D (1996b). Effects of a four-fold increased orthodontic force magnitude on tooth movement and root resorptions. An intra-individual study in adolescents. *Eur J Orthod* 18: 287-294.

- PAGE RC, SCHROEDER HE (1981). Spontaneous chronic periodontitis in adult dogs. *J Periodontol* 52: 60-73.
- QUINN R, YOSHIKAWA D (1985). A reassessment of force magnitude in orthodontics. *Am J Orthod* 88: 252-260.
- REED BE, POLSON AM, SUBTELNY JD (1985). Long-term periodontal status of teeth moved into extraction sites. *Am J Orthod* 88: 203-208.
- REITAN K (1957). Some factors determining the evaluation of forces in orthodontics. *Am J Orthod* 43: 32-45.
- REITAN K (1967). Clinical and histological observations on tooth movement during and after orthodontic treatment. *Am J Orthod* 53: 721-745.
- REITAN K (1969). Principles of retention and avoidance of posttreatment relapse. *Am J Orthod* 55: 776-790.
- REITAN K (1974). Initial tissue behaviour during apical root resorption. *Angle Orthod* 44: 68-82.
- RICHARDSON ME (1965). The direction of tooth movement subsequent to the extraction of teeth in the rhesus monkey. *Rep Congr Eur Orthod Soc* 41: 133-151.
- RYGH P (1977). Orthodontic root resorptions studied by electron microscopy. *Angle Orthod* 47: 1-16.
- RYGH P, BOWLING K, HOVLANDSDAL L, WILLIAMS S (1986). Activation of the vascular system: a main mediator of periodontal fiber remodeling in orthodontic tooth movement. *Am J Orthod* 89: 453-468.
- SAKOMOTO S, SAKOMOTO M (1985). On the possibility that bone matrix collagen is removed prior to bone mineral during active cell-mediated bone resorption. In: Ornoy A, Harell A, Sela J (eds.) *Current advances in skeletogenesis*. Amsterdam, The Netherlands: Elsevier Science, p. 65.
- SANDY JR, MEGHJI S, HARRIS M, MEIKLE MC, FARNDAL RW (1992). Internal and external signals from mechanically deformed osteoblasts: a unifying hypothesis. In: *The biological mechanisms of tooth movement and craniofacial adaptation*, Davidovitch Z (ed.), Columbus, Ohio (USA): The Ohio State Univ., College of Dentistry, pp. 231-239.
- SHAPIRO E, ROEBER FW, KLEMPNER LS (1979). Orthodontic movement using pulsating force-induced piezoelectricity. *Am J Orthod* 76: 59-66.
- SHARPE W, REED B, SUBTELNY JD, POLSON A (1987). Orthodontic relapse, apical root resorption, and crestal alveolar bone levels. *Am J Orthod Dentofac Orthop* 91: 252-258.

- STARK TM, SINCLAIR PM (1987). Effects of pulsed magnetic fields on orthodontic tooth movement. *Am J Orthod* 91: 91-104.
- STOREY E, SMITH R (1952). Force in orthodontics and its relation to tooth movement. *Austr J Dent* 56: 11-18.
- STOREY E (1955). Bone changes associated with tooth movement: a histological study on the effect of force in the rabbit, guinea pig and rat. *Austr J Dent* 59: 147-161.
- TANNE K, SAKUDA M, BURSTONE CJ (1987). Three-dimensional finite element analysis for stress in the periodontal tissue by orthodontic forces. *Am J Orthod* 92: 499-505.
- TANNE K, NAGATAKI T, INOUE Y, SAKUDA M, BURSTONE CJ (1991). Patterns of initial tooth displacements associated with various root lengths and alveolar bone heights. *Am J Orthod Dentofac Orthop* 100: 66-71.
- TIDY DC (1989). Frictional forces in fixed appliances. *Am J Orthod Dentofac Orthop* 96: 249-254.
- WAINWRIGHT WM (1973). Faciolingual tooth movement: its influence on the root and the cortical plate. *Am J Orthod* 64: 278-302.
- WEINSTEIN S, HAACK DC, LIGHTLE MY, SNYDER BB, ATTAWAY HE (1963). On equilibrium theory of tooth position. *Angle Orthod* 33: 1-26.
- WONG A, REYNOLDS EC, WEST VC (1992). The effect of acetylsalicylic acid on orthodontic tooth movement in the guinea pig. *Am J Orthod Dentofac Orthop* 102: 360-365.
- YAMASAKI K, SHIBATA Y, IMAI S, TANI Y, SHIBASAKI Y, FUKUHARA T (1984). Clinical application of prostaglandin E1 (PGE1) upon orthodontic tooth movement. *Am J Orthod* 85: 508-518.
- YAMASAKI K (1989). Pharmacological control of tooth movement. In: *The biology of tooth movement*, Norton LA, Burstone CJ (ed.), Boca Raton, Florida (USA): CRC Press, Inc., pp. 287-320.

Chapter 8

Summary

The correction of malposed teeth is one of the major goals in the treatment of orthodontic patients. The elimination of crowding and spacing, the correction of rotations and abnormal tooth positions, and the alignment of teeth to a proper arch form are restricted to the dento-alveolar part of the maxillofacial complex. An effective orthodontic therapy aims at producing a maximum amount of tooth movement with as little damage as possible to the root, periodontal ligament, and alveolar bone. The present study deals with the relationship between the magnitude of constant and continuously acting orthodontic forces and the rate of bodily tooth movement. The histologic changes in the periodontal ligament during different phases of tooth movement are studied with light microscopy. Finally, the relapse of tooth movement after treatment is studied when no retention measures are taken.

Chapter 1 deals with clinical, biological and biomechanical considerations, which are important in the study of experimental tooth movement.

Chapter 2 describes the time dependent behaviour of orthodontic elastics tested in different media in vitro. Six different types of elastics were tested under four experimental conditions: in artificial saliva in the dark, in distilled water in the dark, in air in natural daylight, in air in the dark. Force measurements show that, after a great initial loss of 13% of the initial tension, elastics kept in artificial saliva and distilled water can produce almost constant forces for at least three weeks. This means that, if in clinical orthodontics constant forces are preferred, there is no rationale for daily renewal of elastics, if they are used with a static loading pattern. Because a large variation was found in initial force levels between elastics of the same type, they should always be measured if force values are critical. When elastics are kept in natural daylight, the force decay is significantly larger than when they are kept in the dark.

Chapter 3 describes the spontaneous migrations of teeth in the mandible of beagle dogs after extraction of the mandibular third premolars. In humans, teeth adjacent to an extraction site, move towards each other and the extraction space is normally reduced. In our experimental group however, a significant increase of most of the interdental distances was found. This unexpected finding may be explained by the diverging eruption pattern of the

mandibular teeth, especially the canine, which has a mesial inclination. Growth of the mandible may provide the additional space required for the mandibular teeth to spread out. Furthermore the tongue may play a role, especially if it fills up the space at the extraction site.

Chapter 4 describes the process of bodily orthodontic tooth movement in young adult male beagle dogs during a period of 112 days. The mandibular second premolars were moved distally with elastics exerting forces of 50, 100, or 200 cN. Tooth movement was measured twice a week with a digital calliper. In the time-displacement curves, four distinct phases could be discerned. No significant differences between the three force groups were found in the duration of each phase, nor in the mean rate of tooth movement during each phase. Large individual differences were found in the rate of orthodontic tooth movement. However, mean rate of tooth movement at the left and the right side, although different forces were used, were highly correlated. Maximum rate of tooth movement was about 2.5 mm per month in all force groups. It was concluded that not the magnitude of the orthodontic forces that were used, but individual characteristics are decisive in determining the rate of orthodontic tooth movement.

Chapter 5 describes the relapse directly after active orthodontic tooth movement during 112 days. There was no period of retention and the orthodontic appliances were left in place. Time-displacement curves showed a rapid initial relapse, followed by a gradual decrease in the rate of relapse to a final stable position. The mean amount of relapse was about 40% of the tooth movement produced before. Mean duration of the relapse period was 78 days. No significant differences between the force groups were found in mean amount of relapse and mean duration of relapse. Significant positive correlations between the amount of active tooth movement on the one hand, and the amount of relapse and the duration of the relapse period on the other hand were found. The mean amount of relapse of the right and the left side was significantly correlated.

Chapter 6 describes the histologic changes in the periodontal ligament and alveolar bone during bodily orthodontic tooth movement and subsequent relapse in beagle dogs. At the pressure side of the periodontal ligament, normal tissue structure was lost soon after the start of the experiment. This

was followed by undermining resorption in the hyalinized areas. Later, only direct osteoclastic activity was found, which was limited to continuously changing local bony protrusions. Root resorptions were present in all force groups, initially as small local spots, which increased with time to extensive resorptions, especially in the middle part of the root. At the tension side osteoid was deposited around newly formed collagenous fibres within 7 days. This was followed by the deposition of trabecular bone in finger-like bony protrusions, oriented in the direction of the stretched fibres. A cementum layer with increasing thickness was deposited along the root surface. Histologic changes in the periodontal ligament were independent of force magnitude. Root resorption seemed to increase with the amount and duration of tooth movement. During relapse after some time reversed cellular activity was observed at the pressure and tension sides and even root resorption was seen at the former tension side. When teeth had come to a standstill, there was still cellular activity to reorganize the tissue structure in the periodontal ligament.

In **Chapter 7** the results of the previous chapters are discussed. Compared to tipping orthodontic tooth movement, bodily tooth movement offers advantages with respect to stress distribution, biologic reactions and tissue damage in the periodontal ligament. Moreover experimental bodily tooth movement offers experimental results that are reproducible and can be a basis for extrapolation to future research. The optimal force theory and the hypothesis that a linear relationship exists between the stress in the periodontal ligament and rate of orthodontic tooth movement are rejected by the present findings. It seems that the discussion on optimizing biomechanical therapy should not concentrate on changing force magnitude and stress levels in the periodontal ligament, but on increasing metabolic activity and cellular processes that might be responsible for the large inter-individual differences in rate of orthodontic tooth movement. The time-displacement curves of the relapse without retention represent the long term recovery of the periodontal ligament and alveolar bone and suggest visco-elastic properties. The mean relapse of 40% of the active tooth movement stresses the "strength" of the information that is stored somehow in the biologic system. Clinical implications of the present findings include the recognition of large individual

differences in rate of bodily tooth movement, which is independent of force magnitude as used in this experiment. On the basis of the present findings suggestions for further research are given.

Chapter 9

Samenvatting

De correctie van een afwijkende tandstand is een van de belangrijkste doelen bij de behandeling van orthodontische patiënten. In tegenstelling tot gelaatsorthopedische correcties beperken orthodontische correcties zich tot het dento-alveolaire deel van het maxillofaciale complex. Een effectieve orthodontische behandeling is erop gericht een maximale tandverplaatsing te bereiken, met een minimale schade aan wortel, parodontaal ligament, en alveolair bot. Het hier gepresenteerde onderzoek bestudeert de relatie tussen de grootte van constante en continu aanwezige orthodontische krachten en de snelheid van bodily (parallele) tandverplaatsing. De histologische veranderingen in het parodontale ligament gedurende de verschillende fasen van tandverplaatsing worden bestudeerd met lichtmicroscopie. Tenslotte wordt de relapse van tandverplaatsing na de behandeling onderzocht indien geen retentie wordt toegepast.

Hoofdstuk 1 behandelt klinische, biologische en biomechanische overwegingen, die van belang zijn bij het onderzoeken van experimentele tandverplaatsing.

Hoofdstuk 2 beschrijft het tijdsafhankelijke gedrag van orthodontische elastieken, getest in verschillende media in vitro. Zes verschillende soorten elastieken zijn getest onder vier experimentele condities: in kunstspeeksel in het donker, in gedestilleerd water in het donker, in de lucht in natuurlijk daglicht, en in de lucht in het donker. Krachtmetingen tonen aan dat, na een groot initieel verlies van 13% van de oorspronkelijke kracht, elastieken die bewaard zijn in kunstspeeksel of gedestilleerd water, een vrijwel constante kracht kunnen leveren gedurende ten minste drie weken. Dit betekent dat, indien in de klinische orthodontie constante krachten worden verlangd, er geen noodzaak bestaat elastieken, die statisch worden belast, dagelijks te vernieuwen. Omdat er een grote variatie in initiële kracht gevonden werd tussen elastieken van dezelfde soort, zouden deze altijd gemeten moeten worden, indien de gewenste krachtgrootte kritisch is. Als elastieken in daglicht bewaard worden, is het krachtverlies significant groter dan in het donker.

Hoofdstuk 3 beschrijft spontane migraties van tanden in de onderkaak van beagle honden na extractie van de derde onderpremolair. Bij de mens bewegen tanden naast een extractiediasteem spontaan naar elkaar toe,

waardoor de ruimte tussen deze tanden kleiner wordt. In onze experimentele groep werd echter een significante toename gevonden van vrijwel alle interdentale afstanden. Deze onverwachte bevinding zou verklaard kunnen worden door de divergerende eruptierichting van de tanden in de onderkaak, vooral van de hoektand, die een mesiale inclinatie heeft. Groei van de onderkaak zou voor additionele ruimte kunnen zorgen, zodat de tanden zich nog verder kunnen verplaatsen. Mogelijk dat ook de tong een rol speelt, zeker indien deze de ruimte van het extractiediasteem opvult.

Hoofdstuk 4 beschrijft het proces van bodily tandverplaatsing bij jong volwassen beagle honden gedurende een periode van 112 dagen. De tweede onderpremolaire werden naar distaal verplaatst met behulp van elastieken, die een kracht leverden van 50, 100, en 200 cN. De tandposities werden twee keer per week gemeten met een digitale schuifmaat. In de tijdverplaatsingscurves konden vier afzonderlijke fasen onderscheiden worden. Tussen de drie krachtgroepen werden geen significante verschillen gevonden in de duur van elke fase, noch in de gemiddelde snelheid van tandverplaatsing gedurende elke fase. Grote individuele verschillen werden gevonden in de snelheid van orthodontische tandverplaatsing. Voor de gemiddelde snelheid van tandverplaatsing bestond een hoge correlatie tussen de linker- en de rechterkant, ondanks het gebruik van verschillende krachten. De maximale snelheid van tandverplaatsing was ongeveer 2,5 mm per maand in alle krachtgroepen. Er werd geconcludeerd dat niet de grootte van de orthodontische krachten die gebruikt werden maar individuele karakteristieken beslissend zijn voor de snelheid van tandverplaatsing.

Hoofdstuk 5 beschrijft de relapse direct na de actieve orthodontische verplaatsing gedurende 112 dagen. Er werd geen retentie toegepast en de orthodontische apparaten bleven in situ. De tijd-verplaatsingscurves toonden een snelle initiële relapse, gevolgd door een geleidelijke afname in de snelheid van de relapse tot een stabiele positie bereikt werd. Gemiddeld bedroeg de hoeveelheid relapse 40% van de eerder bereikte verplaatsing. De gemiddelde duur van de relapse was 78 dagen. Er werden geen significante verschillen tussen de krachtgroepen gevonden in de gemiddelde hoeveelheid relapse of de duur van de relapse. Significante positieve correlaties werden gevonden tussen enerzijds de hoeveelheid actieve tandverplaatsing, en

anderzijds de hoeveelheid relapse en de duur van de relapse. De gemiddelde hoeveelheid relapse aan de rechter- en linkerkant waren significant positief gecorreleerd.

Hoofdstuk 6 beschrijft de histologische veranderingen in het parodontale ligament en alveolaire bot gedurende bodily orthodontische tandverplaatsing en de daarop volgende relapse in beagle honden. Aan de drukzijde van het parodontale ligament ging de normale weefselstructuur snel na aanvang van het experiment verloren. Dit werd gevolgd door ondermijnende resorptie in de gehyaliniseerde gebieden. Later werd alleen nog directe osteoclastische activiteit gevonden, die beperkt bleef tot de meest prominente en continu van plaats veranderende botrandjes. Wortelresorpties waren in alle krachtgroepen aanwezig, initieel als kleine lokale laesies, later toenemend tot uitgebreide resorpties, vooral in het middelste derde deel van de wortel. Aan de trekzijde werd binnen 7 dagen osteoïed afgezet rond nieuw gevormde collageen vezels. Dit werd gevolgd door de afzetting van trabeculair bot in vingervormige uitstulpingen, met een oriëntatie in de richting van de gestrekte vezels. Een cementlaag met toenemende dikte werd afgezet langs het worteloppervlak. Histologische veranderingen in het parodontale ligament waren onafhankelijk van de krachtgrootte. Wortelresorptie leek toe te nemen met de hoeveelheid en de duur van de tandverplaatsing. Gedurende relapse werd met enige vertraging een omgekeerde cellulaire activiteit gezien aan de druk- en trekzijde, en zelfs wortelresorptie werd waargenomen aan de voormalige trekzijde. Nadat de tanden tot stilstand waren gekomen, was er nog steeds cellulaire activiteit om de weefselstructuur in het parodontale ligament te reorganiseren.

In **Hoofdstuk 7** worden de resultaten van de vorige hoofdstukken besproken. Vergeleken met tippende orthodontische tandverplaatsing biedt bodily tandverplaatsing voordelen met betrekking tot de drukverdeling, biologische reacties en weefselschade in het parodontale ligament. Bovendien biedt experimentele bodily tandverplaatsing resultaten die reproduceerbaar zijn en een basis vormen voor extrapolatie naar toekomstig onderzoek. De "optimale kracht" theorie en de hypothese dat een lineaire relatie bestaat tussen de druk in het parodontale ligament en de snelheid van orthodontische tandverplaatsing worden verworpen door de huidige resultaten. Het lijkt erop

dat de discussie over het optimaliseren van de biomechanische therapie zich niet moet concentreren op het veranderen van krachtgroottes en drukniveaus in het parodontale ligament, maar op het versnellen van metabolische activiteit en cellulaire processen, die verantwoordelijk kunnen zijn voor de grote individuele verschillen in snelheid van tandverplaatsing. De tijd-verplaatsingscurves van de relapse zonder retentie representeren het herstel van parodontaal ligament en alveolair bot op lange termijn en suggereren visco-elastische eigenschappen. De gemiddelde relapse van 40% van de actieve tandverplaatsing benadrukt de "kracht" van de informatie die op een of andere manier is opgeslagen in het biologische systeem. Klinische implicaties van de huidige resultaten bevatten de erkenning van grote individuele verschillen in snelheid van bodily orthodontische tandverplaatsing, die onafhankelijk is van de krachtgroottes zoals die in dit experiment gebruikt zijn. Op basis van deze bevindingen worden aanbevelingen gedaan voor verder onderzoek.

Dankwoord

Gaarne wil ik een aantal mensen bedanken die onmisbaar zijn geweest bij de totstandkoming van dit proefschrift.

In de eerste plaats mijn promotor, Prof.dr. A.M. Kuijpers-Jagtman voor haar inzet en begeleiding vanaf de eerste onderzoeksgedachte tot en met de laatste literatuurlijst.

Mijn co-promotor Dr. J.C. Maltha, die met zijn kritische geest en histologische kennis een essentiële bijdrage heeft geleverd.

Prof.dr. F.P.G.M. van der Linden en Prof.dr. H. Boersma voor de opleiding tot orthodontist en de mogelijkheid binnen hun vakgroep onderzoek te doen.

Dr. M.A. van 't Hof, voor de statistische analyses en zijn constructieve opmerkingen.

Dhr. S.J.A.M. Nottet voor zijn assistentie bij het invoeren en verwerken van data.

Drs. E.H.M. Dijkman voor zijn hulp bij het meetprogramma van de röntgenfoto's.

De heren Th.H.M. Arts, A.J. Peters, en P.H.G. Philipsen van het Centraal Dierenlaboratorium voor de voorbereiding en begeleiding van de metingen bij de proefdieren en het maken van de röntgenfoto's.

Drs. S.T. Kusters, voor zijn samenwerking en hulp tijdens het werk op het dierenlaboratorium.

Dhr. T.W. Willemsen voor het vervaardigen van de gegoten orthodontische apparatuur en de heer M.P.H. Mulder voor zijn hulp bij het vervaardigen van de gipsmodellen.

De heer F. Schoenmaker van de Instrumentele Dienst voor het vervaardigen van de nauwkeurige geleidebusjes.

Drs. P.M.M. Willems en Drs. F.A.H. Verhaegh voor het verrichten van de metingen op gipsmodellen.

Mevr. M.P.A.C. Helmich voor het vervaardigen van de histologische preparaten.

Dhr. L.J.H. Hofman voor zijn hulp bij het verzamelen van de literatuur.

Mevr. J.M.J. Verhoeven, office-manager van de vakgroep Orthodontie, voor de uitwerking van het manuscript.

Curriculum vitae

Johannes Jacobus Gertrudis Maria Pilon werd op 12 juni 1959 geboren te Geleen. In 1977 behaalde hij aan de scholengemeenschap St. Michiel te Geleen het diploma Gymnasium-B. Vervolgens studeerde hij een jaar technische natuurkunde aan de Technische Hogeschool in Eindhoven. In 1978 begon hij met de studie tandheelkunde aan de Katholieke Universiteit Nijmegen. In 1982 werd het doctoraalexamen afgelegd en in 1983 het tandartsexamen. Daarna vestigde hij zich als tandarts algemeen practicus in Geleen, waar hij werkzaam is geweest tot dat in 1985 gestart werd met de opleiding tot specialist in de dento-maxillaire orthopaedie aan de Katholieke Universiteit Nijmegen. Sinds 1989 is hij gevestigd als orthodontist in Veldhoven.

Stellingen

behorende bij het proefschrift
Orthodontic forces and tooth movement

J.J.G.M. Pilon
1 oktober 1996

1. Bodily orthodontische tandverplaatsing verloopt volgens een karakteristiek patroon waarin vier stadia te onderscheiden zijn die respectievelijk gekenmerkt worden door: initiële verplaatsing, stilstand, versnelling, en constante verplaatsingssnelheid (dit proefschrift).
2. Onder de omstandigheden als in dit proefschrift beschreven bestaat er geen lineaire dosis-response relatie tussen de grootte van een orthodontische kracht en de snelheid van bodily tandverplaatsing (dit proefschrift).
3. Het gebruik van gelijke orthodontische krachten leidt in beagle honden tot grote individuele verschillen in de snelheid van bodily tandverplaatsing (dit proefschrift).
4. De maximale snelheid van bodily tandverplaatsing in het hier gepresenteerde onderzoek is ongeveer 2,5 mm per maand en is onafhankelijk van de grootte van de gebruikte orthodontische kracht (dit proefschrift).
5. Tijdens de fasen van versnelling en constante verplaatsingssnelheid treedt bij beagle honden geen ondermijnende botresorptie op maar alleen directe resorptie, die strikt gelokaliseerd is op de meest vooruitstekende botspiculae (dit proefschrift).
6. De relapse na een bodily tandverplaatsing gedurende 4 maanden zonder een retentieperiode bedraagt bij beagle honden gemiddeld 40% van de actieve verplaatsing en wordt gekenmerkt door een snelle initiële verplaatsing, gevolgd door een geleidelijke afname in snelheid tot een stabiele positie is bereikt (dit proefschrift).
7. Bij bodily tandverplaatsing is wortelresorptie onvermijdelijk, niet alleen tijdens de actieve verplaatsing maar ook tijdens de relapse (dit proefschrift).

8. Het plaatsen van een spalk achter het onderfront aan het einde van een orthodontische behandeling lost het probleem van recidiverend ruimtegebrek niet op maar verplaatst het naar een ander deel van de tandboog.
9. Het grote probleem bij de overgang van een activator naar vaste apparatuur is het handhaven van de bereikte kaakrelatie, omdat deze vaak gedeeltelijk een tijdelijke spier- en/of ligament-bepaalde houdings-correctie van de onderkaak blijkt te zijn.
10. Bij het levelen van een diepe curve van Spee in de onderkaak wordt het onderfront vaak geprotrudeerd. De parodontale effecten hiervan op lange termijn zijn onvoldoende onderzocht.
11. Use your past successes as a trampoline, not as an easy chair. (Life's little instruction book, volume II)
12. De uitslag van de aanduiding "dermatologisch getest" van lichaamsverzorgende produkten vindt u eerder op uw huid dan op de verpakking.
13. Goede wijn behoeft misschien geen krans, maar verdient er wel degelijk een.

